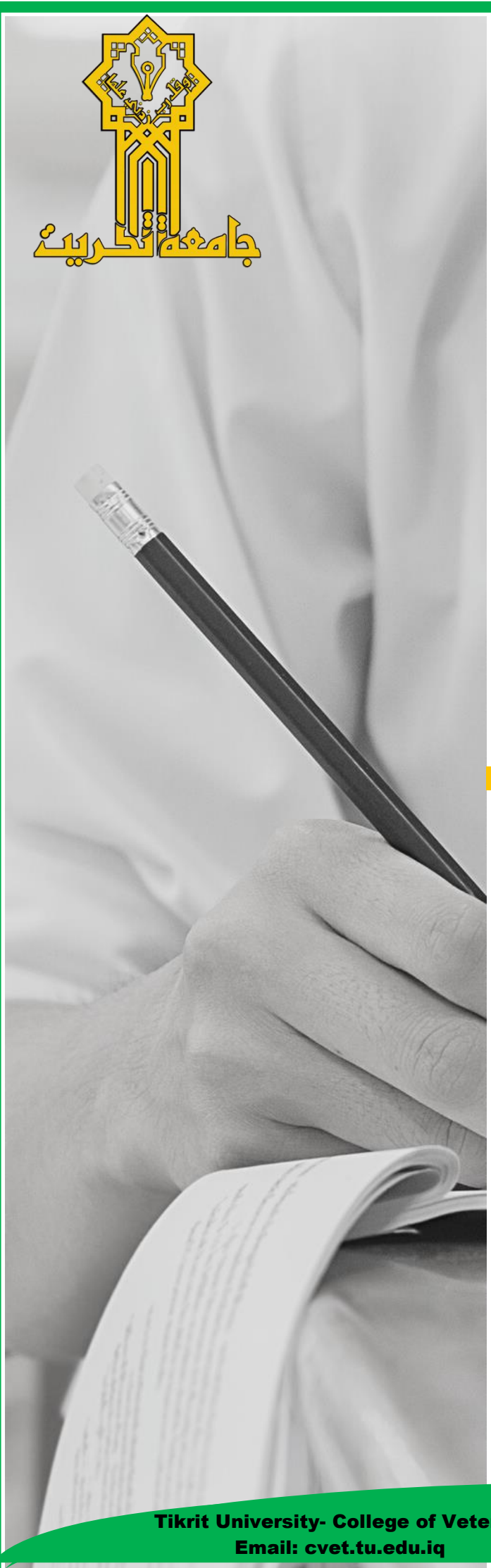

Tikrit University
College of Veterinary Medicine

Diseased Caused By Nutritional Deficiencies

Subject name: internal medicine

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Lecturers link

Selenium and or Vitamin E Deficiencies

❖ **The term vitamin E describes a family of 8 antioxidants**:

- 4 tocopherols ($\alpha, \beta, \gamma, \& \delta$) and 4 tocotrienols.

*** a-** tocopherol is the active form of vitamin E in the human and animal body.

❖ **Function of vitamin E**

- The main function of vitamin E is antioxidant. It intercepts **free radicals** يعترض الجذور الحرة & prevents destruction of cell membrane.
- It protects the **fat in LDL** (**low-density lipoprotein**) from oxidation.
- It inhibits **platelets aggregation**.
- It enhances **يعزز vasodilatation**

❖ Several diseases of farm animals are associated with a deficiency of either selenium or vitamin E alone or in combination, usually in association with predisposing factors such as:

1- Dietary polyunsaturated fatty acids
2- Unaccustomed(Abnormal) exercise
3- Rapid growth in young animals.

❖ The disease occur in selenium and vitamin E deficiency it is called **responsive diseases** because, with some exceptions, they can be prevented by adequate supplementation of the diet with both nutrients.

***Selenium** is a biochemical component of the **enzyme glutathione peroxidase (GSH-PX).** The activity of the enzyme in erythrocytes{ act as intracellular antioxidant role and closely similar to the functions of Vit. E (a-Tocopherol) }. It is positively related to the blood concentration of selenium in cattle, sheep, horses, and pigs and is a useful aid for the diagnosis of selenium deficiency and to determine the selenium status of the tissues of these animals.

***Selenium** is also a component of **thyroid gland hormones.**
* **The variations in GSH –PX enzyme** activity between certain tissues, such as liver, heart, skeletal, and myocardial muscles, would explain the variations in the severity of lesions between species.

Etiology

1- Diets deficient in selenium and/or vitamin E, with or without the presence of conditioning factors.

2- Excessive quantity of polyunsaturated fatty acids in the diet. It may cause Vit. E deficiency because It act as an antioxidant.

3- In the case of naturally occurring muscular dystrophy in calves, lambs and foals on pasture, the myopathic agent, if any, is unknown

4- Interaction between selenium and trace mineral may occur EX. Excessive quantities of cobalt, silver, tellurium, zinc, and vanadium.

***Note**: Selenium – Vet. E supplementation can provide protection against cobalt –induced cardiomyopathy.

Epidemiology

I-Enzootic Nutrition Muscular Dystrophy

1-It occur occurs in all farm animal species, but most commonly in young, rapidly growing calves, lambs, goat kids, and foals born from dams that have been fed for long periods, on diet low Selenium – Vet. E usually during the winter months.

2- Several factors influence the availability of soil selenium to plants Including:

A-Soil PH, alkalinity encourages selenium absorption by plants

B- The presence of a high level of sulfur, which competes for absorption sites with selenium in both plants and animals.

C- Variation between plants in their ability to absorb selenium, EX. selector' and 'converter' plants are listed under the heading of selenium poisoning; legumes take up much less selenium than do grasses.

D-Sedimentary rocks are richer in selenium so plant grown in such areas contain high selenium.

E- Seasonal conditions also influence the selenium content of pasture, the content being lowest in the spring and when rainfall is heavy. Heavily fertilized with superphosphate, thus increasing its sulfate content, if the rainfall is heavy.

3-Cattle and sheep fed on deficient plant in deficient soil cause certain degree of stress and deficient disease.

4- Vitamin E deficiency occurs most commonly when animals are fed inferior quality hay or straw or root crops. While, cereal grains, green pasture, and well-cured fresh hay contain adequate amounts of the vitamin, EX. a-Tocopherol levels are high in green grasses and clovers, but there are wide variations in the concentrations from one area to another.

5- Excessive amount of Selenium may cause selenium poisoning.

6- Muscular dystrophy with myoglubinuria seen in yearling cattle occur due to feeding on high moisture grain which was treated with propionic acid.

7-Vit. E deficiency may occur in calves fed on milk replacer (Diet containing Polyunsaturated fatty acids- P U FAs) resulting Muscular dystrophy, EX. cod liver oil, other fish oils, fishmeal used as a protein concentrate, lard, linseed oil, soybean, and com oils.

8- The condition (NMD) may also occur due to presence of myopathic agents in diet (Unsaturated fatty acids in fish and vegetable oils may be myopathic agents in some outbreaks of NMD of calves (White muscle disease) and lambs (Stiff-limb disease).

9-Outbreaks also reported in lambs and calves during transportation for long distance.

10-Nutritional muscular dystrophy occur in horse most commonly in foals about 7 months of age.

11- Out breaks of disease occur due to animals fed on plants containing gynogenic glycosides.

II- Selenium –Responsive Unthriftiness

This occur in sheep and cattle cause sever economic loss by decrease growth rate, decrease wool growth and chronic diarrhea in calves. All which are improved by selenium supplementation

III- Reproductive performance

- The deficiency of Vit. E and Selenium effect on reproductive performance.
- Selenium deficiency was responsible for decrease infertility and or decrease of production in sheep.

- The supplementation of selenium to ewes (Low selenium status) did not improve reproductive performance.
- Recent Experimental studies using selenium-deficient diets in ewes have been unable to find any adverse effects of selenium depletion on ewe conception rates, embryonic mortality, or numbers of lambs born. But A high incidence (more than 10%) of retained fetal membranes has been associated with marginal levels of plasma selenium compared with herds without a problem.

Pathogenesis

1- Dietary selenium, and Vit. E act as antioxidant protecting tissue from oxidation damage.
2- GsH-PX has major role in detoxifying lipid peroxides by reducing them to non-toxic hydroxyl fatty acids.
3- Vit E prevent formation of Fatty acid hydroperoxidase.
4- High dietary unsaturated fatty acid increase requirements for Vit. E.
5- In adequate level of selenium in diet resulting tissue oxidation occur resulting degeneration and necrosis of cell.
6- Vit. E protect cellular membranes from lipoperoxidation specially membranes rich in unsaturated lipids such as(mitochondria, endoplasmic reticulum and plasma membranes).
7- Diet deficient Vit. E and or selenium lead to permit widespread tissue lipoperoxidation leading to hyaline degeneration and retention of calcium in muscle fibers cause calcification of muscle fiber.
8- Unaccustomed exercise can accelerate oxidative damage and appearance of clinical signs of deficiency.
9- The muscle degeneration cause release of enzymes such as Lactate dehydrogenase (LD), aldolase, and creatine phosphokinase(CPK).
10- In calves , Lambs and foals the major Muscle involvement are:
- Skeletal M. (more common in older animals, weakness, and recombancy)
- Myocardial M. (Acute heart failure)
- Diaphragmatic M.(Respiratory distress, rapid death of animal in spite of treatment)

11- Acute Muscular dystrophy resulting in the liberation of Myoglobinuria (more common in horses, older calves and yearling cattle which have lower concentration of Myoglobin in their muscles).

Clinical signs (Clinical finding)

I-Acute enzootic muscular dystrophy

1-Affected animals may collapse and die suddenly after exercise without any other premonitory signs specially after exercise (effected young calves and foals).

2-Sudden onset of dullness and severe respiratory distress, accompanied by a frothy or bloodstained nasal discharge.

3-Lateral recumbency and may be unable to assume sternal recumbency even when assisted.

4-The heart rate is usually increased up to 150-200/min and often with arrhythmia, the respiratory rate is increased up to 60-72/min and loud breath sounds are audible over the entire lung fields.

5-The temperature is usually normal or slightly elevated.

6-Affected animals commonly die 6-12 h after the onset of signs in spite of therapy.

7-Outbreaks of the disease occur in calves and lambs in which up to 15 % of susceptible animals and the case fatality approaches 100%.

II-Subacute enzootic muscular dystrophy

- This is the most common form in rapidly growing calves, 'white muscle disease'
- In young lambs, 'stiff-limb disease'.

1- Affected animals may be found in sternal recumbency and unable to stand but some make an attempt to stand.
2- Stiffness, trembling of the limbs, weakness and termer
3- In most cases, an inability to stand for more than a few minutes.
4- Abnormal gait
5-Bilaterally swollen and firmer than normal of dorso-lumber , gluteal and shoulder muscle.
6-dyspnea with labored and abdominal-type respiration. Due to of skeletal and diaphragmatic muscle are effected.
7- Transient fever (41°C, 105°F) due to the effects of myoglobinemia and pain.
8-Increase the heart rate
8-The affected animals usually respond in a few days and within 3-5 days they are able to stand and walk unassisted.

III-Paralytic myoglubinuria

1- It occur in yearling cattle usually following moving from in indoor to outdoor pasture in about 9-18 mouth of age.
2-Clinical signs as in above except myoglubinuria may be present.

VI-Subclinical nutritional muscular dystrophy

1-Occurs in apparently normal animals in herds at the time clinical cases are present.
2-The serum levels of creatine phosphokinase levels may be elevated in susceptible
animals for several days before the onset of clinical signs; f
3-Treatment with vitamin E and selenium the level of serum enzymes returns to normal.
4- Grossly abnormal electrocardiograms occur in some animals and may be detectable before clinical signs are evident.

V-Congenital muscular dystrophy

1- It has been described in a newborn calves
2-The calf was still recumbent 13 h after birth, had increased serum creatine kinase and decreased serum vitamin E and selenium levels.
3-Recovery occurred following supportive therapy and vitamin E and selenium.

Clinical Pathology

1-Plasma creatine kinase (C K) is the most commonly used laboratory aid in the diagnosis of NMD. This enzyme is highly specific for cardiac and skeletal muscle and is released into the blood following unaccustomed exercise and myodegeneration.

2-Aspartate aminotransferase (AST) activity is also an indicator of muscle damage,
but is not as reliable as the CK because increased AST levels may also indicate liver damage.

3-Selenium status:

a- Estimation of selenium levels in soil and plants is accumulating gradually, the estimations are difficult and expensive. Most field diagnoses are made on the basis of clinicopathological findings.
b- The response to treatment and control procedures using selenium.

c- GHS-PX analyses, direct relationship between activity of this enzyme and level of selenium in blood and tissues of animal.
d- Estimation of selenium concentration in animal, blood, and milk.

4-Estimation of GHS-PX in blood and tissues of animal.

5-Vit. E status

a- Vit. E occur in nature as mixture of alpha-tocopherol, bata-tocopherol, gama-tocopherol, delta-tocopherol and other tocopherols in varying proportion.
b- Estimation of tocopherol level in blood and liver provide good formation on Vit. E status of the animal. But difficult of the laboratory assay of tocopherols they are not commonly done.

Necropsy finding

1- Bilaterally symmetrical and contain localized white or gray areas of degeneration and necrosis with the appearance of fish flesh in skeletal, myocardial, and diaphragmatic muscles.
2- The affected muscle is friable and edematous and may be calcified
3- Signs of pneumonia due to secondary infection
4- Cardiac hypertrophy and pulmonary congestion, edema and calcification of lesions has been observed.
5- Histologically the muscle lesions are non-inflammatory, hyaline degeneration is followed by coagulation necrosis.

Diagnosis

1- Clinical finding 2- lab. Investigation 3- Clinical pathology

Differential Diagnosis

1- Disease cause Septicemia 2- Pneumonia 3- Toxemia

Treatment

1- Mixture of intramuscular injection containing 3mg selenium (as sodium or potassium selenite) and 150 IU/ml of Dalta-alpha-tocopherol acetate and the dose rate of treatment is 2ml/45 Kg. B.Wt.
2- Vit E and selenium supplementation in to diet as powder.
3- AD3E injection 3ml/50 Kg. B.Wt.

Control

1-Dietary supplementation
2-Individual prophylactic injection and oral administration
3-Successfully prevented of diseases caused by selenium deficiency by administration of selenium to dam during pregnancy and directly to young animal (selenium transported cross the placenta and give protection for the neonate).
4-Pasture topdressing (the application of sodium selenite as a topdressing to pasture is now practiced and permitted in some countries).