**SELENIUM AND OR VITAMIN E DEFICIENCIES**

**Etiology**

The selenium- and vitamin E-responsive or deficiency diseases of farm animals are caused by diets deficient in selenium and/or vitamin E, with or without the presence of conditioning factors such as an excessive quantity of polyunsaturated fatty acids in the diet, , unaccustomed exercise and rapid growth in young animal.

**Biological functions of selenium and vitamin E**

Glutathione peroxidases and tissue peroxidation Selenium is a biochemical component of

the enzyme glutathione peroxidase (GSH-PX). The activity of the enzyme in erythrocytes is positively related to the blood concentration of selenium in cattle, sheep, horses, and pigs and is a useful 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.

Plasma GSH-PX protects cellular membranes and lipid- containing organelles from peroxidative damage by inhibition and destruction of endogenous peroxides, acting in conjunction with vitamin E to maintain integrity of these membranes.

Selenium also facilitates significant changes in the metabolism of many drugs .

For example.

selenium functions to counteract the toxicity of several metals such as arsenic, cadmium, mercury, copper, silver, and lead.

The Selenium important in, phagocytosis, all the immune response and are important mediators of immune and reproductive function.

**Vitamin E**

Vitamin E is an antioxidant that prevents oxidative damage to sensitive membrane lipids by decreasing hydroperoxide formation.

The vitamin has a central role in protection of cellular membranes from lipoperoxidation, especially membranes rich in unsaturated lipids, such as mitochondria, endoplasmic reticulum, and plasma membranes. Interrelationships between selenium and vitamin E

An important interrelationship exists between selenium, vitamin E and the sulfur-containing amino acids in preventing some of the nutritional diseases caused by

their deficiency.

**EPIDEMIOLOGY**

**Enzootic nutritional muscular dystrophy (NMD)**

**Occurrence**

This muscular dystrophy 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, usually during the winter months, on diets low in

selenium and vitamin E It is an important cause of mortality in goat kids from

birth to about 3 months of age.

The disease is not well recognized in adult horses, but sporadic cases of dystrophic myodegeneration are recorded in horses from 5 to 10 years of age.

The disease also occurs in grain-fed yearling cattle

Outbreaks of severe and fatal NMD have occurred in heifers, at the time of parturition, which were previously on a diet deficient in both selenium and vitamin E.

Myopathy and hepatic lipidosis in weaned lambs deficient in vitamin E without concurrent selenium deficiency has been described.

There are two major syndromes of myopathy:

1- An acute form: myocardial dystrophy, which occurs most commonly in young calves and lambs and occasionally foals

2- A sub acute form: skeletal muscular dystrophy, which occurs in older calves and yearling cattle.

**PATHOGENESIS**

The antioxidant roles of selenium and vitamin E. Dietary selenium, sulfur containing amino acids and vitamin E act synergistically to protect tissues from oxidative damage which is selenium-dependent, functions by detoxifying lipid peroxides and reducing them

to non- toxic hydroxy fatty acids.

Vitamin E prevents fatty acid hydroperoxide formation. vitamin E protects cellular membranes from lipoperoxidation, especially membranes rich in unsaturated lipids, such as mitochondric, endoplasmic reticulum and plasma membranes.

Diets low in selenium and/or vitamin E do not provide sufficient protection against the

'physiological' lipoperoxidation that occurs normally at the cellular level.

Selenium has a sparing effect on vitamin E and is an efficient prophylactic against musculardystrophy of calves and lambs at pasture, but does not prevent muscular dystrophyin calves fed on a diet containing cod liver oil.

**Nutritional muscular dystrophy:-**

Diets deficient in selenium and/or vitamin E permit widespread tissue lipoperoxidation

leading to hyaline degeneration and calcification of muscle fibers.

Unaccustomed exercise can accelerate the oxidative process and precipitate clinical

disease. Muscle degeneration led to the release of enzymes, such as lactate dehydrogenase and creatine phosphokinase, the last of which is of importance in diagnosis.

Degeneration of skeletal muscle is rapidly and successively followed by invasion of phagocytes and regeneration.

In calves, lambs, and foals, the major muscles involved are skeletal, myocardial

and diaphragmatic.

The myocardial and diaphragmatic forms of the disease occur most commonly in young calves, lambs, and foals, resulting in acute heart failure, respiratory distress, and rapid death.

The skeletal form of the disease occurs more commonly in older calves, yearling cattle, and older foals and results in weakness and recumbence, is usually less severe and responds to treatment.

Acute NMD results in the liberation of myoglobin into the blood, which results

in myoglobin urea.

**CLINICAL FINDINGS**

**Acute enzootic muscular dystrophy:-**

1 - Affected animals may collapse and die suddenly after exercise without any other

premonitory signs.

2 - The excitement associated with the hand-feeding of dairy calves may precipitate peracute death.

3 – In calves under close observation, a sudden onset of dullness and severe respiratory

distress, accompanied by a frothy or bloodstained nasal discharge, may be observed

in some cases.

4 - Affected calves, lambs, and foals are usually in lateral recumb ency and may be unable to sternal recumbency .

5 - The heart rate is usually increased up to 200/min and often with arrhythmia, the respiratory rate is increased up to 60-72/min and loud breath sounds are audible. 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 . may develop the acute form and the case fatality approaches 100%.

**Sub acute enzootic muscular dystrophy:-**

1- The most common form in rapidly growing calves, 'white muscle disease' and

in young lambs, 'stiff-lamb disease'.

2- Affected animals may be found in sternal recumbence and unable to stand ,If they are

standing, the obvious signs are stiffness, trembling of the limbs, weakness.

3-The gait in calves is accompanied by rotating movements of the hocks and in lambs a stiff, goosestepping gait.

4- Muscle tremor is evident if the animal is forced to stand for more than a few minutes. 5-5-On palpation the dorsolumbar, gluteal and shoulder muscle masses may be symmetrically enlarged and firmer than normal.

6- Most affected animals retain their appetite and will suck if held up to the dam or eat if hand-fed.

7- Major involvement of the diaphragm and intercostal muscles causes dyspnea with labored and abdominal-type respiration.

8-The temperature in the normal range but there may be a transient fever (41°C, 105°F)

due to the effects of myoglobinemia and pain. The heart rate may be elevated .

9- Following treatment, affected animals usually respond in a few days and within

3-5 days they are able to stand and walk unassisted.

10-In some cases, the upper borders of the scapulae protrude above the vertebral column and are widely separated from the thorax. This has been called the'flying

scapula' .

11- the toes are spread and there is relaxation of carpal and metacarpal joints or knuckling at the fetlocks and standing on tip-toe, inability to raise the head, difficulty in swallowing inability to use the tongue and relaxation of abdominal muscles.

12- Choking may occur when the animals attempt to drink.

13-Clinical signs occur within 1 week and consist of stiffness, recumbency, myoglobinuria, hyperpnea, and dyspnea. Severe cases may die within a few days and some are found dead without premonitory signs.

**Subcapsular liver rupture in lambs**

has been associated with vitamin E deficiency in lambs usually under 4 weeks of

age , Affected lambs collapse suddenly, become limp, and die within a few

minutes or several hours after the onset of weakness.

**Clinical pathology** Increased plasma levels of creatine kinase. Low serum levels

of selenium and vitamin E. Glutathione peroxidase activity.

**Necropsy findings** Bilaterally symmetrical pale skeletal muscle, pale streaks in myocardial muscle. Hyaline degeneration of affected muscle. Diagnostic confirmation Low selenium and vitamin E in diet and tissues, increased creatine kinase and muscle

degeneration.

**Differential diagnosis list**

Acute muscular dystrophy in calves and yearlings:

• Haemophilus somnus septicemia

• Pneumonia.

Subacute enzootic muscular dystrophy:

• Musculoskeletal diseases

polyarthritis, traumatic or infectious myopathies (blackleg), osteodystrophy,

and fractures of long bones

Diseases of the nervous system:

spinal cord compression, Haemophilus somnus meningoencephalitis and myelitis, organophosphatic insecticide poisoning

Diseases of the digestive tract:

carbohydrate engorgement resulting in lactic acidosis, shock, dehydration and weakness.

• Muscular dystrophy in lambs and

kids: Enzootic ataxia and swayback

**Treatment** Vitamin E selenium

parenterally. it is recommended that a combined mixture of selenium and IX-tocopherol be used in treatment.

**Nutritional muscular dystrophy**

For treatment of NMD in calves, lambs, and foals a mixture containing 3 mg selenium (as sodium or potassium selenite) and 150 IU/mL of DL-IX-tocopherol acetate, given 1M at 2 mLl45 kg BW is recommended. One treatment is usually

sufficient.

Animals with severe myocardial involvement will usually not respond to treatment and the case mortality rate is about 9 0 % .

**Control** Selenium and vitamin E supplementation of diet, strategic oral and/or parenteral vitamin E and selenium to pregnant dams or young animals on pasture.