Selenium Toxicosis in Veterinary Medicine

Dr Mostafa Abdollahi

DVSc of Large Animal Internal Medicine

Graduated from University of Tehran (Iran)

Assistant Professor: Faculty of Veterinary Medicine, Semnan University, Semnan, Iran

Email: mostafaabdollahi@semnan.ac.ir

Key points

•**Dual-Nature Element:** Selenium is an essential component of the enzyme glutathione peroxidase (GSH-Px) and thyroid hormones, but it has a **very narrow margin of safety**.

•**Biological Requirement:** Approximately 0.1 to 0.3 mg/kg (ppm) of the dry matter (DM) in the diet.

•**Toxicity Threshold:** Dietary concentrations exceeding 2 to 5 mg/kg can be toxic (the margin between requirement and toxicity is sometimes only 10-fold!).

•**Economic and Clinical Significance:** Induces severe hoof and epithelial lesions, abortion, and acute pulmonary mortality in ruminants and equids.

Sources and Etiology of Selenium Toxicity

1.Obligate / Primary Accumulators:

•These plants strictly require soils rich in selenium for growth and can accumulate thousands of ppm of selenium.

•Representative species: Selenium-accumulator milk vetches (*Astragalus* spp.), Prince's plume (*Stanleya pinnata*), and woody asters (*Oonopsis*).

2.Facultative / Secondary Accumulators:

•They can grow without selenium, but in seleniferous soils, they store high concentrations (several hundred ppm), such as *Aster* and *Atriplex* species.

3.Iatrogenic / Management-Related Factors:

•Errors in calculating mineral supplements or premixes in feed mills.

•Dosage errors when injecting selenium + vitamin E preparations (especially in lambs, calves, or horses, rapidly leading to acute toxicity and sudden death).

•Toxicity resulting from drinking water or industrial effluents rich in selenium.

Selenium Toxicosis in Iran

•**Geological Deficiencies in Iran:** The majority of Iranian soils are selenium-deficient; the primary clinical challenge in domestic livestock remains **selenium deficiency** (e.g., White Muscle Disease, reproductive failure) rather than toxicity.

•**Absence of Obligate Accumulators:** Unlike certain regions globally (e.g., North America), native *Astragalus* species and pastures in Iran lack obligate selenium accumulation capabilities; natural selenosis from forage is virtually non-existent.

•**Iatrogenic Etiology of Toxicity:** Isolated cases of selenium toxicosis in Iranian livestock are strictly management-related, resulting from **incorrect dosages, repeated injections of Se-Vitamin E preparations**, or feed-mill premix formulation errors.

Pathogenesis

•**Selenium-Sulfur Substitution:** Selenium incorporates into sulfur-containing amino acids (methionine and cysteine) instead of sulfur, producing non-functional proteins (such as selenoproteins altering normal structure) that severely damage tissues with high metabolic activity and rapid cellular turnover.

•**Oxidative Stress:** At high doses, instead of acting as an antioxidant, selenium generates reactive oxygen species (ROS), causing damage to cell membranes and mitochondria.

•**Target Tissue Systems:** Liver, kidneys, and epithelial/keratinized tissues (e.g., hooves, hair, and skin).

Acute Selenium Toxicosis: Clinical Manifestations

1.Common Causes: Usually results from miscalculated injections of selenium + vitamin E supplements, oral drenching errors, or sudden access to primary (obligate) accumulator plants.

2.Onset & Course: Very rapid (from a few hours up to 24–48 hours post-exposure).

3.Key Clinical Signs:

•**Respiratory/Cardiovascular:** Severe dyspnea, rapid labored breathing (tachypnea), frothy discharge from the nose/mouth due to severe acute pulmonary edema, and acute cardiovascular collapse.

•**Gastrointestinal:** Severe abdominal pain (colic), watery diarrhea, anorexia, and excessive salivation.

•**Neuromuscular:** Extreme weakness, muscle tremors, inability to stand (recumbency), progressing to coma and sudden death.

4.Characteristic Odor: A garlic-like or radish-like odor on the animal's breath, resulting from the pulmonary excretion of dimethyl selenide.



Chronic Selenium Toxicosis (Alkali Disease)

•**Etiology and Occurrence:** Chronic consumption of forage or diets containing excessive selenium concentrations (approximately **5–40 mg/kg of feed**) over weeks to months.

•**Damage to Keratinized Tissues:** Selenium substitution for sulfur in keratin disrupts the structure of hooves and hair, making them brittle.

•**Characteristic Clinical Signs:**

•**Hair loss and alopecia:** Loss of coarse guard hairs; in horses, marked loss of the mane and tail (“bobtail” appearance); and reduced fleece density in sheep.

•**Hoof lesions and deformities:** Circumferential horizontal cracks in the hoof wall below the coronary band, severe lameness, abnormal hoof growth and deformity, and, in advanced cases, sloughing of the hoof capsule.

•**Progressive weight loss and emaciation**, with impaired growth due to hoof pain and difficulty grazing or walking.

•**Reproductive effects:** Reduced fertility, lower conception rates, and abortion.











Blind Staggers Syndrome

•**Historical View:** Traditionally described as a subacute form of selenium poisoning in ruminants grazing on primary (obligate) accumulator plants (*Astragalus* spp.).

•**Clinical Progression Stages:**

•**Stage 1:** Wandering, separation from the herd, reduced visual acuity, anorexia, and inability to navigate obstacles.

•**Stage 2:** Apparent blindness, head pressing against solid objects, severe limb weakness, and excessive salivation.

•**Stage 3:** Pharyngeal and esophageal paralysis (inability to swallow), severe muscle spasms and tremors, recumbency, and ultimately death due to respiratory failure.

•**Current Scientific Consensus:**

•Research has demonstrated that pure selenium alone does not reproduce the classic neuropathological lesions of "Blind Staggers."

•The majority of these clinical signs are actually caused by **Polioencephalomalacia (PEM)** resulting from high dietary sulfates or co-occurring toxic nitrogenous/alkaloidal compounds (such as miserotoxin and indolizidine alkaloids) found in *Astragalus* plants, rather than selenium toxicity alone.

Necropsy & Pathological Findings

- **Acute Lesions:**
- **Massive Pulmonary Edema & Congestion:** Lungs are heavy, congested, and filled with frothy fluid, accompanied by hydrothorax and hydropericardium.
- **Petechial and Ecchymotic Hemorrhages:** Present on the pericardium, endocardium, epicardium, and gastrointestinal mucosa.
- **Acute Hepatic and Renal Necrosis:** Pallor, centrilobular hepatic congestion, and acute tubular necrosis in the kidneys.
- **Garlic/Radish Odor:** Distinctly noticeable upon opening the abdominal and thoracic cavities.

- **Chronic Lesions:**
- **Hoof Wall and Epithelial Changes:** Separation of the hoof wall from the laminar tissue, hyperkeratosis, abnormal growth, and severe deformity.
- **Hepatic Interstitial Fibrosis and Cirrhosis:** Liver becomes firm, shrunken, and dark yellowish-brown.
- **Erosion and Necrosis of Articular Cartilages:** Particularly in the stifle, hock, and fetlock joints, leading to degenerative joint disease (arthropathy).
- **Cardiac Muscle Atrophy and Chronic Hydropericardium.**

Diagnostic Approaches & Differential Diagnosis

- **Sample Matrices & Laboratory Confirmation:**
- **Acute Toxicity:** Whole blood, liver, and kidneys (highest accumulation sites). Liver selenium concentrations exceeding **4–5 mg/kg (wet weight)** indicate toxicity.
- **Chronic Toxicity:** Sampling of **hair (mane and tail)** and **hoof wall** (rich in keratin, reflecting long-term exposure; values above **5–10 mg/kg** are pathognomonic).
- **Environmental Analysis:** Testing selenium levels in drinking water, forage, and total mixed rations (TMR).

Paraclinical Findings

Marked elevation in liver enzymes (AST, SDH, GGT) and renal markers (BUN, creatinine) due to tissue necrosis.

Differential Diagnosis

•**Acute Form:** Organophosphate poisoning, nitrate/nitrite toxicity, hypomagnesemia (sudden death/tetany), and enterotoxemia.

•**Chronic Form:** Laminitis, ergotism, fluorosis (skeletal and hoof changes), and alopecias due to nutritional deficiencies or ectoparasites.

•**“Blind Staggers”:** Thiamine-responsive polioencephalomalacia (PEM), lead poisoning, and listeriosis.

Treatment (Acute form)

•**Lack of Specific Antidote:** There is no specific chemical antidote available to directly neutralize selenium within tissues.

•**Supportive & Shock Therapy:** Oxygen therapy, management of acute pulmonary edema (diuretics with caution due to renal failure), precise IV fluid therapy, and corticosteroids to combat cardiovascular collapse.

•**Gastrointestinal Decontamination:** Activated charcoal and gastric lavage if ingestion occurred very recently, to bind inorganic selenium compounds.

Treatment (Chronic form)

•Immediate removal of the contaminated feed source or supplement and relocation from seleniferous pastures.

•**Sulfur-Selenium Competition:** Supplementing the diet with non-toxic sulfates (e.g., sodium sulfate) or proteins rich in sulfur-containing amino acids (methionine/cysteine); sulfates reduce both intestinal absorption and renal tubular reabsorption of selenium.

•**Hoof and Pain Care:** Corrective hoof trimming, soft bedding, and non-steroidal anti-inflammatory drugs (NSAIDs) such as flunixin meglumine or meloxicam to alleviate lameness pain.

Prevention Strategies

•Extreme precision in calculating doses and diluting injectable selenium + vitamin E preparations.

•Regular forage, TMR, and water testing in regions with seleniferous soils.

•Pasture management: Preventing overgrazing and controlling selenium-accumulator plants (such as *Astragalus* spp.).