Molybdenum Toxicosis / Molybdenosis

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

•**Essential trace element with a critical Copper relationship:** Molybdenum functions as a cofactor in enzymes such as xanthine oxidase, yet its major clinical significance lies in a **marked antagonism toward copper** within the gastrointestinal tract.

•**Core condition (Molybdenosis):** Manifested primarily as **secondary copper deficiency (hypocuprosis)**, despite adequate dietary copper intake.

•**Main susceptible species:** Ruminants, particularly dairy cattle and growing calves (greatest sensitivity), followed by sheep.

•**Economic impact:** Chronic diarrhea, progressive weight loss, coat discoloration (a “silver-grey” or “bleached” haircoat), reproductive impairment, and reduced productivity.

Sources & Etiology of Molybdenum Toxicosis

•High-Molybdenum Soils & Peat Soils:

While molybdenum is immobilized in acidic soils, **alkaline and poorly drained/wet soils** markedly increase plant uptake of Mo.

Pastures grown on peat/muck soils containing elevated Mo are historically associated with severe enteritis syndromes (**"Teart" pastures / Peat Scours**).

Forage Accumulation (Legumes vs. Grasses):

Legumes (especially alfalfa and clovers) accumulate molybdenum at significantly higher concentrations than grasses.

•Industrial & Mining Contamination:

Aerial deposition of industrial dusts from steel-smelting plants, copper/molybdenum mines, alloy manufacturing facilities, and oil refineries contaminating adjacent pastures.



•**Agricultural Practices:**

Over-application of molybdenum-containing fertilizers (used to stimulate legume nodulation) and land application of industrial/municipal sewage sludge.

•**Dietary Copper-to-Molybdenum Ratio (Cu:Mo):**

Toxicity depends strongly on relative proportions rather than absolute Mo levels alone; when the dietary Cu:Mo ratio drops below **2:1 or 3:1** (ideal is 6:1 to 10:1), molybdenosis inevitably occurs.

Pathogenesis of Molybdenum Toxicosis

•**Rumen Thiomolybdate Formation:** Dietary molybdate, in the presence of sulfur, is converted by rumen microorganisms into thiomolybdates.

•**Copper Binding:** Thiomolybdates bind firmly to copper, forming insoluble, poorly absorbed complexes that drastically reduce copper absorption from the gastrointestinal tract.

•**Secondary Copper Deficiency:** The sharp decline in copper absorption and bioavailability disrupts copper-dependent enzymatic functions, leading to functional copper deficiency despite adequate dietary copper levels.

•**Systemic Copper Disruption:** Certain thiomolybdates can be absorbed into the bloodstream, binding systemic copper and further impairing cuproenzyme activities (e.g., cytochrome c oxidase, ceruloplasmin) in target tissues.

•**Role of Sulfur & Mineral Ratios:** The severity of toxicity heavily depends on dietary sulfur and the precise Cu:Mo ratio, making absolute molybdenum measurements alone insufficient for risk assessment.

Susceptible Species

Cattle (especially dairy cows and growing calves) are highly sensitive; sheep are somewhat more resistant but still exhibit secondary copper deficiency signs. Equine and porcine species are remarkably resistant due to the absence of a rumen where thiomolybdates are formed.

Clinical Signs

•Persistent Watery Diarrhea (“Peat Scours” / “Teart”):

The hallmark early sign is profuse, foul-smelling, bubbly, green-to-black watery diarrhea that fails to respond to conventional antidiarrheal treatments.

•Coat Depigmentation & Achromotrichia:

Caused by inhibition of the copper-dependent enzyme tyrosinase, impairing melanin synthesis.

Black hair coats turn dull brown, grey, or reddish; characteristic “spectacle eyes” (light-colored hair rings around the eyes) frequently develop.

In sheep, loss of wool crimp and a straight, harsh texture known as **“steely wool”**.

•Bone, Joint & Lameness Disorders:

Inhibition of lysyl oxidase (reducing collagen and elastin cross-linking) leads to osteoporosis, epiphyseal enlargement, joint stiffness, and spontaneous fractures.

•Productivity & Reproductive Failures:

Severe emaciation, sharp drops in milk production, silent heats, delayed puberty, and poor conception rates.

Pathological Findings & Necropsy Lesions

•**Gastrointestinal Lesions:**

•Severe catarrhal or hemorrhagic enteritis and abomasitis.

•Villous atrophy and mucosal edema throughout the small intestine, secondary to chronic profuse diarrhea and severe malabsorption.

•**Skeletal & Cartilaginous Lesions:**

•Thinning of cortical bone and generalized osteoporosis/osteopenia.

•Widening and enlargement of the epiphyseal growth plates (resembling rickets), accompanied by articular cartilage erosions and joint stiffness.

- **Hematological & Vascular Changes:**

  Microcytic hypochromic anemia resulting from impaired ferroxidase (ceruloplasmin) activity, which disrupts iron mobilization from tissue reserves.

  Weakness and structural fragility of large blood vessel walls (such as the aorta) due to defective elastin cross-linking.

- **Central Nervous System (Enzootic Ataxia / Swayback):**

  In newborn lambs from ewes exposed to high molybdenum and secondary copper deficiency: demyelination of the spinal cord and brainstem, alongside motor neuron necrosis leading to hindlimb ataxia.

Diagnosis

•**Copper & Molybdenum Assays:** Quantifying copper and molybdenum concentrations in serum, plasma, and liver tissue (liver is the gold standard for assessing body copper status; hepatic copper levels below 20–25 ppm dry weight indicate deficiency).

•**Dietary Cu:Mo Ratio:** Analyzing forage and water samples to confirm a dietary copper-to-molybdenum ratio of less than 2:1.

•**Biochemical Markers:** Evaluating the activity of copper-dependent enzymes such as serum ceruloplasmin and erythrocyte copper-zinc superoxide dismutase (Cu/Zn-SOD).

Treatment & Nutritional Correction

•**Copper Supplementation:** Subcutaneous or intramuscular administration of copper compounds (e.g., Copper calcium EDTA) with extreme caution to prevent acute copper toxicity, particularly in sheep.

•**Dietary Fortification:** Supplementing copper sulfate directly into the feed or drinking water to overcome the antagonistic effects of excess molybdenum.

•**Managing Dietary Sulfates:** Monitoring and limiting dietary sulfate levels to prevent the enhanced ruminal formation of thiomolybdates.

Prevention

•Managing grazing in alkaline, peaty, or mineral-contaminated pastures ("teart" soils) and limiting the intake of legume-dominant forages known to accumulate molybdenum.

•Routine monitoring of soil, water, and forage mineral profiles, especially in volcanic, mining, or alkaline geological zones.

Nature of Occurrenc in Iran

In most regions of Iran, primary high-dose molybdenum toxicosis is rare; instead, the predominant clinical presentation in sheep, goat, and cattle herds is **secondary copper deficiency (hypocuprosis)** induced by antagonistic interactions of molybdenum, sulfur, and iron in soil, forage, and water.

Role of Alkaline & Calcareous Soils

A vast portion of Iran’s central plateau and arid/semi-arid zones features **alkaline and calcareous soils (pH>7.5)**, which enhance forage molybdenum uptake while impairing plant copper acquisition, drastically lowering the dietary copper-to-molybdenum (Cu:Mo) ratio.

Nutritional & Management Factors in Iranian Livestock in Iran

•**High Alfalfa (Lucerne) Intake:** Alfalfa, serving as the staple forage in Iranian farming systems, acts as a potent molybdenum accumulator compared to grasses.

•**High-Sulfate Well Waters:** Subsurface borehole waters across central and southern provinces contain elevated sulfate levels; the combination of forage molybdenum and water sulfates synergistically promotes ruminal thiomolybdate formation and severe copper blockade.

Regional Research Evidence in Iran

•Documented field studies (e.g., in Khuzestan, West/East Azerbaijan, and Zagros regions) have reported cases of **enzootic ataxia (Swayback)** in newborn lambs secondary to elevated forage molybdenum levels.

•Investigations in Khuzestan province (Ramhormoz, Masjed-Soleyman, Behbahan) confirmed that simultaneous antagonistic actions of molybdenum, high iron, and sulfur in pasture forages push small ruminants into borderline or clinical copper deficiency.

Dominant Clinical Picture in Iranian Flocks

Achromotrichia (hair/wool depigmentation and browning), “steely wool”, chronic treatment-refractory diarrhea, and developmental skeletal/neurological defects in newborn lambs.