

TOXICITY OF MOLYBDENUM (Mo)

Molybdenum is an essential element associated with a variety of metalloenzymes and corresponding metabolic functions. It is an essential trace element and a component of number of enzymes. Excessive dietary intake of molybdenum induces a secondary copper deficiency. The syndrome, predominately reported in ruminants (versus non-ruminant species) is seen worldwide. Cattle and sheep are 10-fold more susceptible than other species. Horses and Pigs not usually affected. Acute toxicity associated with massive doses is rarely encountered.

SOURCES OF TOXICITY

The interactions associated with copper, molybdenum, and sulfate metabolism related to the utilization, bioavailability, and kinetics of copper are among the most biologically significant interrelationships in veterinary medicine.

1. Excessive use of Mo containing fertilizers may occasionally result in high Mo content in soil and forages.
2. Grazing of animals on pastures with high Mo leads to toxicity of Mo.
3. Ingestion of plants rich in Mo but low in copper contents.
4. Calves may be poisoned by milk from cows on high Mo diets.

TYPES OF MOLYBDENUM TOXICITY

There are two types of molybdenum toxicity. The first, which is well recognised, occurs on high molybdenum pastures (often referred to as 'teart' pastures) containing around 20 - 100 mg of molybdenum per kg. Within a few days of being placed on such pastures, cattle start scouring and develop stary, harsh coats. This can be effectively prevented by high doses of copper, because it has been demonstrated that such levels of molybdenum affect the absorption and metabolism of copper.

The second type of molybdenum toxicity that at much lower levels of molybdenum (such as <5 mg of molybdenum per kg of feed), problems of toxicity can occur even in animals which are being fed more copper than their calculated dietary requirements and have an apparently normal copper status. It is suggested that in adult cattle affected by this version of molybdenum toxicity, the most common problem reported by the farmer is poor fertility.

MECHANISM OF TOXICITY

1. In ruminants, various molybdates react with sulfides to produce thiomolybdate compounds, which react with copper to form an insoluble complex that is poorly absorbed.
2. The limited bacterial formation of thiomolybdates in monogastric animals is primarily responsible for the tolerance to molybdenum encountered in these species.
3. Excessive molybdenum exposure may also impair a variety of enzymes involved in collagen and elastin maintenance and stability, which has been associated with cardiovascular disorders.
4. Molybdenum exposure may reduce phospholipid synthesis in nervous tissue, resulting in demyelination and neurologic disorders clinically.

Molybdenum toxicity has been encountered in regions of the world containing peat, muck, or shale soil types that are naturally contaminated with molybdenum. Industrial contamination associated with mining or metal production or areas using molybdenum-contaminated fertilizers result in enhanced uptake of molybdenum by plants used as a feed source.

FACTORS AFFECTING TOXICITY

The susceptibility to molybdenum toxicity in ruminants depends on a number of factors including

1. Dietary copper content - susceptibility increases as the copper content decreases
2. Dietary sulfate content -susceptibility increases with high sulfate levels by impairing copper utilization, whereas low sulfate content enhances susceptibility by reducing molybdenum excretion
3. Chemical form of molybdenum—water-soluble forms found in fresh feed are more toxic
4. sulfur-containing amino acids may alter copper utilization or molybdenum excretion
5. Animal species - cattle are more susceptible
6. Age - young animals are more susceptible, and excretion of molybdenum into milk may produce toxicoses in nursing calves
7. Season of year - molybdenum concentrations in plants increase in the fall
8. Plant species - legumes bioaccumulate more molybdenum
9. Breed differences - seen in sheep and goats.

TOXICITY

Mo is present in all biological fluids and tissues including bones. Mo has a strong inverse interaction with copper and sulphur. Molybdenosis usually results from inadequate Cu: Mo ratio in soil, fodder and diet. In the diet, Cu: Mo ratio of

= 6:1 – ideal

= 2:1 to 3:1 – borderline

= less than 2:1 – toxic

Ratios exceeding 15:1 may cause chronic copper poisoning. Absolute molybdenum concentrations in the diet >10 mg/kg will cause poisoning independent of

copper consumption. Massive molybdenum exposure in the ration >2,000 mg/kg will result in death of the animals.

CLINICAL FINDINGS

Mo toxicity in animals is commonly called as Molybdenosis or Teart. The manifestations of molybdenum toxicity are related primarily to impaired copper metabolism and utilization, resulting in secondary copper deficiency. Typically, the syndrome is a herd problem, with morbidity as high as 80%.

In cattle, clinical disease is characterized by severe, persistent diarrhea with the **presence of green, liquid feces containing gas bubbles**, often referred to as **peat scours or teart scours**. Depigmentation, resulting in fading achromotrichia of the hair coat, is evident in black animals associated with impaired tyrosinase activity and reduced melanin synthesis. Pica, unthriftiness, microcytic hypochromic anemia, emaciation, joint pain characterized by lameness, and osteoporosis often manifested by bone fractures are seen.

Molybdenum competes with phosphorus utilization, resulting in reduced mineralization of bone. In heifers, fertility is reduced. Delayed puberty, poor conception rates, decreased weight at puberty, and decreased milk production are common. Reduced libido has been reported in bulls.

In sheep, particularly in lambs <30 days old, the animals exhibit stiffness of the back and legs and have difficulty rising. The syndrome in sheep is known as **“Enzootic ataxia” in Australia or “Swayback” in U.K.** Abnormal development of connective tissue and growth plates are apparent in affected animals. Manifestations appear within 1–2 wk if molybdenum levels are excessive.

Occasionally, acute toxicity may be encountered in cattle or sheep. Anorexia and lethargy may be evident within 3 days. Deaths begin within 1 wk and may continue for many months. Neonates frequently exhibit hind limb ataxia that often progresses to the forelimbs. Salivation and scant mucoid feces are common.

At necropsy, hemosiderosis, periacinar to severe hepatic necrosis, and nephrosis are evident. In affected sheep, neuronal degeneration, demyelination, and lysis of white matter are seen in nervous tissue.

DIAGNOSIS:

Distinguishing between primary copper deficiency and secondary copper deficiency related to excessive molybdenum exposure is important. Often with molybdenum toxicity, there is a poor correlation between tissue concentrations of copper and clinical disease. Clinical improvement after copper sulfate administration provides valuable support for the diagnosis.

Analysis of the ration for copper and molybdenum concentrations is recommended. Analysis of the liver and plasma for molybdenum provides useful insight to confirm the diagnosis, but the concentrations must be interpreted in association with the comparable tissue concentrations of copper. In cattle, liver concentrations >2 ppm (wet weight) and plasma concentrations >0.1 ppm are consistent with a diagnosis of molybdenum poisoning in the presence of a low copper status.

Other disease syndromes characterized by

- emaciation or unthriftiness (parasite infections, selenosis, fluorosis, ergotism),
- diarrhea (metal poisonings, GI infections),
- lameness or bone abnormalities (fluorosis, selenosis, ergotism, lead poisoning)

may resemble molybdenum poisoning and should be investigated as possible etiologies.

TREATMENT AND PREVENTION:

- Most treatment options are associated with the biological interactions associated with copper, molybdenum, and sulfate.

- Under circumstances in which dietary exposure is difficult to eliminate, simple treatment with copper products may be futile. If the source of molybdenum can be removed, excess molybdenum is rapidly eliminated and food products are safe for consumption within a relatively short time.
 - If the dietary exposure cannot be reduced, elimination of molybdenum in the milk may produce toxicosis in nursing calves.
 - Dietary supplementation with copper sulfate will reduce the bioavailability of molybdenum in the GI tract, ultimately reducing absorption and enhancing excretion.
 - In feed containing molybdenum at >5 mg/kg, supplementation with 1% copper sulfate in salt will control development of the syndrome.
 - In recovered mining areas, the supplementation may need to be increased to as much as 5% copper sulfate in the salt.
 - When the consumption of mineral supplements is impractical, the treatment may be administered as a weekly drench.
 - Injectable products such as copper glycinate or copper edetate (Cu-EDTA) may be given at a dose of 120 mg/cow. These products are approved only in some jurisdictions.
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