Arsenic Toxicosis

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

Definition & Significance

•Arsenic is a highly toxic metalloid and a well-recognized environmental contaminant, historically notorious as a classic poison in both intentional and accidental intoxications of livestock and humans.

•Its veterinary toxicological significance stems primarily from acute, often fatal intoxications arising from herbicides, insecticides, **rodenticides**, livestock dips, and industrial or mining sources.

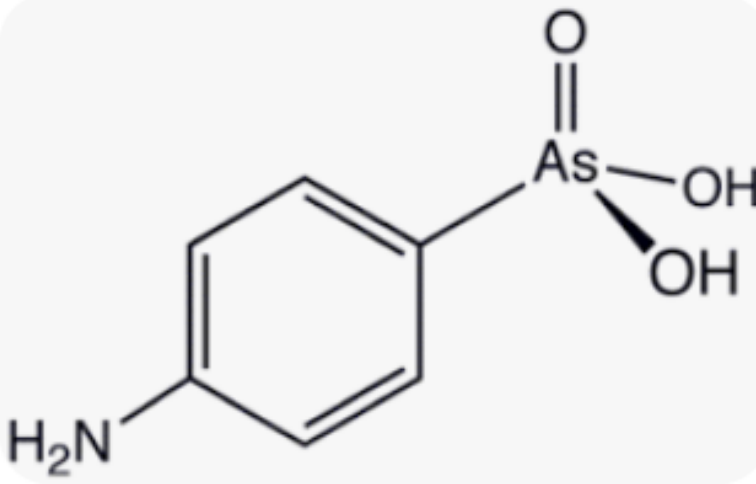
Chemical Forms & Relative Toxicity

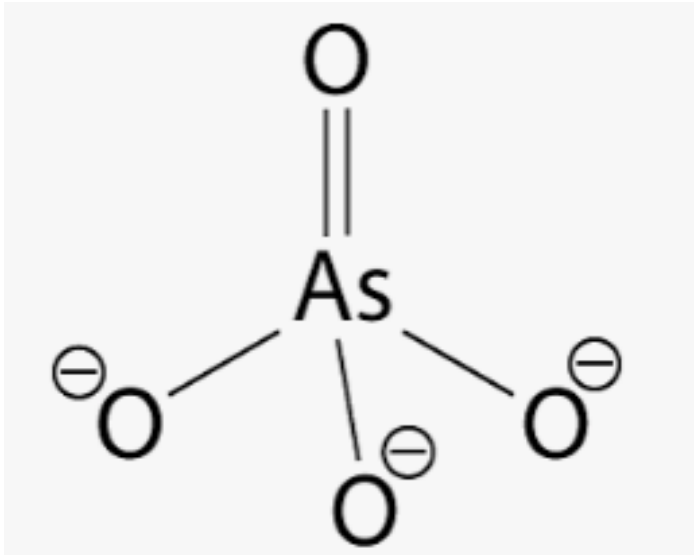
•**Inorganic Arsenic:** Trivalent (Arsenite, As3+) and pentavalent (Arsenate, As5+) compounds.

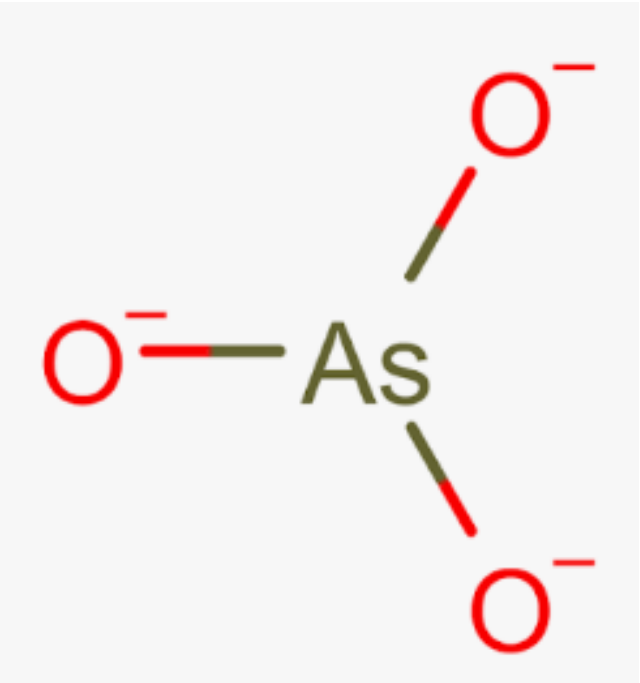
•Arsenites (trivalent): the most toxic forms (e.g., arsenic trioxide, sodium arsenite).

•Arsenates (pentavalent): less toxic, yet disrupt metabolism by structurally mimicking phosphate.

•**Organic Arsenic:** Carbon-containing forms of markedly lower toxicity (e.g., arsanilic acid and poultry growth-promoting feed additives).


O
As
OH
OH
H2N


O
As
O
O
O


O
O
As
O

Sources & Routes of Exposure

•**Environmental & Geogenic:** Soils and waters of volcanic zones, mineral-rich sediments, and arseniferous rocks.

•**Agricultural:** Herbicides (desiccants), insecticides, **rodenticides**, and chromium-copper-arsenate (CCA) wood preservatives.

•**Industrial:** Flue dust and fumes from **metal smelters**, **coal combustion**, and **electronic waste**.

•**Livestock Feed:** Contaminated bran and forage, drinking water, and geophagia (soil-licking) over mineralized pastures.










GROVE
Porous
Volcanic Rocks





Sources

•**Access to Agricultural Toxins:** Improper storage, disposal, or application of arsenical herbicides (e.g., desiccants), insecticides, and rodenticides in agricultural fields and storage facilities.

•**Obsolete Livestock Practices:** Historical use of arsenical livestock dips and legacy organic arsenical feed additives (**such as roxarsone in poultry**) previously used for growth promotion.

•**Treated Timber:** Wooden structures or fencing treated with Chromated Copper Arsenate (CCA) preservatives, which may be chewed or licked by livestock (geophagia).

Geogenic & Environmental Factors: Volcanic zones, thermal hot springs, metalliferous deposits, and mine tailings (representing major sources of natural arsenic in groundwater and surface water).

Industrial contamination and waste runoff surrounding metal smelters and manufacturing plants.

•**Species & Age:** Ruminants (especially cattle) are highly susceptible to acute inorganic arsenicals.

•**Solubility & Chemical Form:** Trivalent arsenites (As3+) are significantly more toxic than pentavalent arsenates (As5+); soluble powders and liquid preparations are absorbed most rapidly.

•**Nutritional Status:** Deficiencies in sulfur-containing amino acids (methionine, cysteine) increase cellular vulnerability to arsenic-induced oxidative stress.

Pathogenesis of Arsenic Toxicosis

•Binding to Sulfhydryl Groups:

Trivalent arsenic (arsenite, As^{3+}) binds to sulfhydryl (–SH) groups in proteins and enzymes, impairing their activity. Energy-metabolism enzymes, particularly the pyruvate dehydrogenase complex, are important targets.

•Disruption of Energy Metabolism:

Pentavalent arsenic (arsenate, As^{5+}) is structurally similar to phosphate and can substitute for it in phosphate dependent reactions. This reduces ATP production and disrupts oxidative phosphorylation.

•Vascular and Gastrointestinal Injury:

Injury to capillary endothelium increases vascular permeability, causing fluid and protein loss, edema, hypotension, and shock. The gastrointestinal mucosa is also highly susceptible and may develop severe inflammation and hemorrhage.

•Oxidative Stress and Organ Injury:

Reactive oxygen species (ROS) and impaired antioxidant defenses contribute to damage to cellular membranes and organelles. The liver and kidneys are among the major organs affected.

Clinical Signs of Arsenic Toxicosis

•Acute Toxicosis:

Sudden onset, profound weakness, lethargy, and prostration (downer animal).

Severe watery diarrhea (frequently containing blood or necrotic mucosal shreds), colic, and severe abdominal pain.

Rapid hypotension, tachycardia, weak pulse, cold extremities, and death secondary to hypovolemic shock.

Excessive salivation, nausea, and vomiting (in susceptible species).

•Chronic Toxicosis:

Anorexia, progressive emaciation (wasting), and general weakness.

Dry and rough coat, alopecia, hoof brittleness, and keratinization abnormalities.

Anemia, pale mucous membranes, and decreased production performance.

Chronic hepatic and renal injury accompanied by mild neurological signs in some cases.

Pathological & Necropsy Findings in Arsenic Toxicosis

•Gross Lesions:

Severe congestion and edema of the gastrointestinal mucosa, especially the abomasum and small intestine.

Severe congested gastritis/enteritis with watery, hemorrhagic, or mucoid contents.

Congestion and degeneration of the liver, kidneys, lungs, and heart secondary to vascular permeability and shock.

•Histopathological Lesions:

Acute necrosis and degeneration of the intestinal and abomasal mucosa with epithelial destruction and sloughing.

Hepatocyte necrosis and renal tubular degeneration (tubular necrosis).

Serous effusions, pulmonary edema, and petechial hemorrhages in multiple organs.

Diagnostic Consideration

In acute cases, arsenic toxicosis may grossly resemble acute infections and enterotoxemias; differentiation requires toxicological and confirmatory laboratory testing.

Diagnosis

•History of exposure to arsenical compounds (herbicides, rodenticides, CCA-treated timber) with acute onset of signs.

•Clinical signs and gastrointestinal/vascular pathological lesions.

•Measurement of arsenic concentration in the liver and kidneys (target organs) and ruminal/gastric contents; elevated tissue arsenic levels confirm the diagnosis.

•Appropriate sample collection (liver, kidney, urine, gastrointestinal contents) and submission to a toxicology laboratory.

Treatment

•**Classic adjunct:** Sodium thiosulfate, administered as a partial antidote to reduce arsenic absorption.

•**Alternative approach:** Dimercaprol (BAL: 2 until 4 mg/kg/4h; Deep IM), followed by succimer (DMSA: PO; no standard dose in animals, 100-500 mg/human/8hr), as chelating agents that enhance arsenic excretion.

•Supportive care: Intravenous fluid therapy to correct hypovolemic shock and electrolytes, control of abdominal pain, and hepatorenal protection


DIMERSYL
Dimercaprol Injection 50 mg/m
DIMERSYL
For IM use only
farbe firose


DIMSA
1x10 Capsules
Livsuccimer
200 mg
Succimer Capsules

Prevention

•Safe storage and keeping of herbicides, pesticides, and toxic baits out of reach of animals.

•Keeping livestock away from arsenic-treated (CCA) timber and contaminated mining/industrial sites.

•Regular monitoring of water and forage sources in high-risk geogenic areas and periodic feed testing.

Status of Arsenic Toxicosis in Iran

•**Historical Trend:** Generalized arsenic poisoning in Iranian livestock is now rare and largely accidental; arsenical compounds have been largely discontinued as feed additives (e.g., roxarsone in poultry) in recent years.

•**Common Causes:**

•Accidental exposure to pesticides, herbicides, and rodenticide baits on farms and in storage facilities.

•Chewing or licking of CCA-treated timber in old structures and pens.

•**Geogenic Exposure:**

•Regions with naturally arsenic-containing soils and water sources (particularly in the Alborz and Zagros provinces with volcanic and metallogenic geology) may contain arsenic in livestock drinking water.

•Groundwater in areas such as the Azerbaijan and Kurdistan provinces is under potential arsenic monitoring, but acute livestock poisoning from this route is rare.

•**Field Reality:** In practice, acute arsenic toxicosis is very rarely encountered in Iranian veterinary medicine, and most cases are mistaken for other acute gastrointestinal toxicoses (such as enterotoxemia); a definitive diagnosis requires toxicological laboratory testing.