ARSENIC TOXICITY/POISONING

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Sources

1. Arsenic poisoning in animals is caused by inorganic and organic arsenical compounds.
2. Arsenite (As+3) is more toxic than arsenate (As+5).
3. Solubility, species of animal involved, and duration of exposure affect poisoning.
4. Inorganic Arsenicals –
    a) These include arsenic trioxide, arsenic pentoxide, sodium and. potassium arsenate, sodium and potassium arsenite, and lead or calcium arsenate.
    b) The lethal oral dose of sodium arsenite in most species is from 1-25 mg/kg. Cats may be more sensitive.
    c) Arsenites are used to some extent as dips for tick control
    d) Lead arsenate is sometimes used as a taeniacide in sheep.
5. Organic arsenicals-
    a) Monosodium methanearsonate (MSMA) and disodium methanearsonate (DSMA) accumulate in plants cause arsenic poisoning, in grazing animals.
    b) Thiacetarsamide and arsphenamine for the treatment of adult heartworms in dogs, phenylarsonic acids and their salt used as feed additives to improve production in swine and poultry rations and also to treat dysentery in pigs.
5. The high arsenic concentration can be attributed to contamination of water from industrial activities and pesticides fodder and water. Drinking water containing >0. 25% arsenic is considered potentially toxic, especially for large animals.

Mechanism of arsenic toxicity

1. Trivalent form of arsenic is thought to be the cause of intoxication.
2. Arsenic acts on and binds sulfhydryl groups within cells.
3. Disrupts and inhibits sulfhydryl-containing enzymes of aerobic metabolism.
4. Tissues affected include gastrointestinal tract, kidney, liver, and lungs.

5. Capillary damage , dilation, transudation and decreased blood perfusion to the splanchnic tissues.
6. It results in decreased systemic blood volume and shock

Toxicokinetics

1. Soluble forms of arsenic compounds are well absorbed orally.
2. Most of the arsenic is bound to RBC; it distributes to several tissues, with the highest levels found in liver, kidneys, heart, and lungs.
3. In subchronic or chronic exposures, arsenic accumulates in skin, nails, hooves, sweat glands, and hair.
4. The majority of the absorbed arsenic is excreted in the urine as inorganic arsenic or in methylated form

Clinical Findings

Acute toxicity

1. Severe vomiting, diarrhea, cramps in muscles and injuries in blood vessels.
2. Arsenic cause damage to cappillaries, transudation of plasma, loss of blood, and hypovolemic shock.
3. Profuse watery diarrhea, sometimes tinged with blood.
4. Severe colic, dehydration, weakness, depression, weak pulse, and cardiovascular collapse.
5. In peracute poisoning, animals may simply be found dead.

Chronic toxicity

1. Hyperkeratosis of skin and damages in kidney and liver.

Diagnosis

1. History,clinical signs,PM lesions
2. Chemical determination of arsenic in tissues (liver or kidney) or stomach cohtents provides confirmation. Liver and kidneys of normal animals rarely contain >1 ppm arsenic (wet wt); toxicity is associated with a concentration >3 ppm.
3. PM lesions:

a) Inflammation and reddening of GI mucosa, edema, necrosis of epithelial and subepithelial tissue.

b) GI contents are often fluid, foul smelling,'and blood tinged.
c) Diffuse inflammation of the liver, kidneys, and other visceral organs.
d) The liver may have fatty degeneration and necrosis, and the kidneys have tubular damage.
e) The skin may show necrosis and be dry or leathery.

Treatment

1. Emesis should be induced (in capable species), activated charcoal with a cathartic then oral administration of GI protectants (small animals, 1-2 hr after charcoal) kaolin-pectin, and fluid therapy as needed.
2. Dimercaprol (British antilewisite, 4-7 mg/kg, 1M, tid for 2-3 days or until recovery.
3. In large animals, thioctic acid (lipoic acid or a-lipoic acid) may be used alone (50 mg/ kg, 1M, tid, as a 20% solution) or in combination with dimercaprol (3 mg/kg, 1M, every 4 hr 1- 2 days, 3 times for the third day, and two times for the next 10 days or until recovery
4. Sodium thiosulfate PO, at 20-30 g in 300 mL of water in horses and cattle, O. 5-3 g in small animals, or as a 20% 40 mg/ kg intravenous (IV)for 8 hour.
5. Once paralysis occurs, the nerve damage is irreversible.

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