

**HEAVY METALS POISONING IN ANIMALS**

**“Heavy Metals” are chemical elements with a specific gravity that is at least 5 times the specific gravity of water.**

**e.g Cadmium antimony, arsenic, chromium, cobalt, copper, gallium, gold, iron, lead, mercury, nickel, platinum, silver, tellurium, thallium, aluminum, uranium, and beryllium .**

**In general heavy metals produce their toxicity by forming complexes or “Ligand” with organic compounds.**

**These modified biological molecules lose their ability to function properly ,and result in malfunction or death of the affected cells.**

**The most common groups involved in ligand formation are oxygen ,Sulfur, and nitrogen .**

**When metals bind to these groups they may inactive important enzyme systems ,or affect protein structure.**

**General treatment for metals poisoning :-**

**Chelating therapy :-**

**1-Dimercaprol .(BAL)British anti leuist .**

**2-Ethylenediamine-tetra-acetic acid or EDTA.**

**3-Penicillamine.**

**4-Deferoxamine .**

**5-Succimer .**

# **1-Arsenic Poisoning**

**-Duration of exposure. Sources of poisoning: Poisoning occurs due to arsenic trioxide, arsenic pentoxide, sodium and potassium arsenite.**

**Toxicokinetics:-**

**After absorption, arsenic is distributed throughout the body but tends to accumulate in the liver and kidneys.**

**In domestic animals, arsenic does not stay in soft tissues for long period.**

**It is rapidly excreted in bile, milk, saliva, sweat, urine and feces.**

**After chronic exposure the poison stays in bones, skin and keratinized tissues. Such as hoof and hair .**

**Arsenic does not cross the blood brain barrier.**

**Milk that is poisoned by arsenic can be toxic to humans.**

**Clinical Signs:**

**Acute: Profuse diarrhoea, severe colic, dehydration, weakness, depression, weak pulse, cardiovascular collapse.**

**Peracute: Animals are found dead.**

**Subacute cases: The animal may live for several days signs include colic, anorexia, depression, staggering, weakness, diarrhea, with blood and mucosal shreds.**

**Chronic: Are rare and are characterized by wasting, poor condition, thirst, rich –red mucosal membrane, normal temperature and a weak and irregular pulse.**

**Differential diagnosis:-**

**Caustic poisoning**

**Irritant paint poisoning**

**Urea chlorate**

**Pesticides poisoning**

**Lead poisoning**

**Treatment:-**

**Administration of G.I protectants e.g. Activated charcoal, kaolin-pectin.**

**Supportive fluid therapy.**

**Administration of BAL (Dimercaprol) at 4-5mg/kg, deep intramuscular.**

**D-Penicillamine 10-50mg/kg, orally.**

## **2-Lead Poisoning**

**In Veterinary Medicine, lead is one of the most common causes of metallic poisoning in dogs and cattle.**

**Only 1-2% of the ingested lead may be absorbed. The organic form of lead could penetrate intact skin. Organic forms are tetraethyle lead and tetramethyl lead.**

**Sources of poisoning:-**

**Animals may ingest lead-based paints.**

**Lead tetraoxide, carbonate, or sulphate**

**Engine oil and lead battery improperly disposed**

**Animals feed agents sprayed with lead insecticides (lead arsenate).**

**Toxicokinetics:-**

**Lead salts are sparingly soluble**

**Absorption of lead from GIT is very limited (1-2%) and therefore about 98% of lead is eliminated in faeces.**

### **Clinical Signs:**

**In cattle, GIT and nervous signs after 24hours of exposure to toxicity  
blindness, salivation, spastic twitching of eyelid, jaw champing, tremor  
convulsion.**

### **Differential diagnosis:**

**Polioencephalomalacia.**

**Tetanus.**

**Hypovitaminosis A.**

**Hypomagnesemic tetany.**

**Insecticide poisoning.**

### **Treatment**

**Magnesium sulphate .**

**Barbiturates tranquilizer may be useful to control convulsion.**

**Calcium disodium edentate given intravenously or subcutaneously at  
110mg/kg bw for three days .**

**D-Penicillamine can be administered orally at 110 mg/kg/day for two  
weeks.**

**Calcium borogluconate I.V recommended at 250-500mg/kg.**

### **3-Mercury poisoning :**

**Mercury can combine with a methyl group to become methyl mercury .  
this form found in environmental pollution and produce toxicities.**

**1-Acute toxicity .**

**2-Chronic toxicity .**

**In acute toxicity: affect GIT , break down barriers in capillaries, and neurological toxicities .**

**In chronic toxicity:CNS affect ,bone ,jaw,teeth,liver and kidney.**

**Treatment:-**

**EDTA and Penicillamine.**

## **4-Cadmium poisoning :**

**Mechanism of toxicity .**

**Free cadmium binds to protein sulfhydryl groups ,disrupting the cellular redox cycle ,depleting glutathione ,and eliciting intracellular oxidant damage .**

**Toxicokinetic .**

**Concentrations of cadmium in liver and kidney but muscle ,and bone do not accumulate high concentrations of cadmium.**

**Blood cadmium concentrations are indicators of recent exposure.**

**Treatment:-**

**Metal chelators and Zinc usefull for protection.**

## **5-Cooper poisoning :**

**Acute poisoning and chronic poisoning .**

**Most animals affected are sheep .**

**Acute poisoning:** is usually seen after accidental administration and cause severe gastroenteritis characterized by abdominal pain, diarrhea, anorexia, dehydration and shock.

Hemolysis and hemoglobinuria may develop after 3 days.

**Chronic poisoning:** is most commonly in sheep when excessive amounts of copper ingested for prolonged period.

**Mechanism of toxicity:**

Blood copper concentrations increase suddenly, causing lipid peroxidation and intravascular hemolysis.

Liver enzymes including ALT and AST are usually increased.

**Treatment :-**

Often, treatment is not successful.

1-Penicillamine.

2-Administration of ammonium molybdate and sodium sulfate.

3-Dietary supplementation with zinc acetate (250 ppm) may be useful to reduce absorption of copper.

## **6-Poisoning by sodium chloride:-**

Salt toxicity (sodium chloride, NaCl), which is more appropriately called sodium ion toxicosis"

can result when excessive quantities of salt are ingested and intake of.

The most sensitive species, cattle, and poultry. Sheep are relatively resistant. The acute oral lethal dose of salt is 2.2 g/kg in swine and 6.0 g/kg in sheep.

**Clinical signs :-**

**In cattle, signs of acute salt poisoning involve the 1- GI tract and 2-CNS. Salivation, increased thirst, vomiting, abdominal pain, and diarrhea are followed by ataxia, circling, blindness, seizures, and partial paralysis.**

**Diagnosis:- Serum and CSF concentrations of sodium  $>160$  mEq/L, especially when CSF has a greater sodium concentration than serum, are indicative of salt poisoning.**

**Differential diagnoses :-include :-**

**1- insecticide poisoning (organochlorine, organophosphorous, and carbamate).**

**2-Lead poisoning especially in cattle .**

**3- polioencephalomalacia, hypomagnesemic tetany, and the nervous form of ketosis.**

**Treatment:- 1-There is no specific treatment. Immediate removal of offending feed or water is imperative.**

**2-Severely affected animals should be given water via stomach tube.**

**The mortality rate:-**

**may be  $>50\%$  in affected animals regardless of treatment.**

**In small animals, slow administration of hypertonic dextrose or isotonic saline may be useful.**