Lecture title: Rodenticides

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Rodenticides

Rodenticides: - are substance that kill rodents, especially mice and rat .

They may classify according to their chemical structure in to:-

1- Inorganic rodenticides :- e.g. zinc phosphide ,

2- Organic rodenticides: - e.g. anticoagulant (warfarin), fluoro acetate .

Inorganic rodenticides: - e.g. 1- zinc phosphide

This agent is used in developing nations because cheap and an effective rodenticides.

Mechanism of toxic action:-

The toxicity of the chemical can be accounted for the phosphine (ph3) formed following a hydrolysis reaction with the stomach on ingestion.

Zinc phosphide + H2O $\longrightarrow$ PH3

Phosphine causes cellular toxicity with necrosis of the GIT and injury to the other organ such as liver and kidney.

Clinical signs of toxicity:-

Vomiting, diarrhea, cyanosis , tacky- cardia , fever and albumin urea, also pulmonary edema and convulsion .

Treatment:-

- In early case ingestion of paraffin oil or coconut oil or NaHCO3
- In symptomatic case : mostly supportive and symptomatic directed to correct shock (Noradrinaline) and metabolic acidosis (NaHCO3).
- IV N-acetylcysteine and vitamin C to decrease oxidative stress .
- Decontamination

Fluoroacetate:-

These compounds are white in color, odorless and tasteless. Ingestion is the major route by which poisoning occurs, with nausea, vomiting and abdominal pain common within 1hr, sweating, apprehension, confusion and agitation follow soon after. Seizures are the main neurological feature, and coma may persist for several days. Fluoroacetate poisoning is associated with citrate accumulation in several tissues, including the brain.

Mechanism of toxic action:-

Inhibition of citric acid cycle, which is part of tricarboxylic acid cycle.

Fluoroacetate inhibit two enzymes in the TCA aconitase (enzyme) and prevents the converted of citric to isocitrate in cycle .

And (ii) succinate dehydrogenase, which catalyzes succinate metabolism. This inhibits TCA cycle .

Inhibition of this system results in reduced glucose metabolism and cellular respiration and affected tissue energy. The most susceptible organs are the heart, kidney and brain.

Clinical signs of toxicity:-

Nausea, vomiting, abdominal pain and tachycardia , hypotension , renal failure and muscle spasms .

C.N.S. signs:- agitation seizures and coma .

Treatment :-

1- No specific anti dotes

2- Glycerol mono acetate proved a beneficial effect in the treatment of poisoned animal .

3-Supportive therapy .

Anticoagulant rodenticides:-

Compound dangerous to all mammals and birds, most are frequent cause poisoning in pets.

Large group of compounds available as pellets, tracking power and baits .intoxication in domestic animals have resulted from combination of feed with anticoagulant concentration and feed mixed in equipment used to prepare rodent baits.

Warfarin:-

Mechanism of action:-

Inhibition synthesis of vitamin K-which essential for normal blood coagulation . they are anticoagulant via interference with clotting factors II, VII,IX and X.

Signs of toxicity:-

In first 12 to 24 hours: No effects noticed

After 12 hours: anticoagulant effect appear in form of :

1- Bleeding from the nose and gums, pain in the joint from hematoma in the long bone, Swollen joints.

2- Pain in the abdomen and back.

3- Weakness from anemia.

4- Pale mucous membranes

5- Shock and death.

6- • Lesions

• Blood in body cavities

• hemorrhage

Diagnosis:-

1- Case history

2- Clinical signs

3- Lab. Test :- citrated blood :-

a- Decrease P.C.V.

b- Prolong activated clotting time (intrinsic pathway).

c- Prolong partial thromboplastin time (intrinsic pathway).

d- Prolong prothrombin time (extrinsic pathway). Clotting time 25% longer than normal suggest poisoning.

4- Normal platelets and fibrin degradation products.

5- Chemical analysis of vomitus, blood and liver.

Treatment:-

1- Gastric decontamination (induces emesis, administer activated charcoal and cathartic).

2- Observe animal closely for clinical signs in the next week.

3- The use of specific antidote vit. K. 3–4 weeks to ensure protection from the longer-acting agents • Monitoring therapy • repeat measurement of prothrombin time 2–5 days after cessation of vitamin K.therapy • improvement in clotting function should occur within 12 hours

4- Blood transfusion is indicated if so plasma concentrations if hemorrhage is sever.

5- Vit. C is occasionally given protect capillaries from damage.

6- O2 therapy if needed.

7- owner education • When sending animal home with vitamin K tablets or capsules, inform owner to continue therapy until clotting test results are normal. • Restrict animal's activity during therapy to prevent trauma. • Observe the animal closely for clinical signs. • Educate owners about proper placement of baits