Lecture title: Herbicides

Lecturer Affiliation: University of Mosul / College of Veterinary Medicine /

Department of Physiology, Biochemistry and Pharmacology

Herbicides: commonly known as weed killers, are substances used to control undesired plants, also known as weeds. or means those chemical that act on the undesirable weeds in almost any crop.

There are two types of herbicides:-

Inorganic herbicides : e.g. arsenicals

Organic herbicides : e.g. phenoxy herbicides .

Arsenicals (Na+ - arsenite and arsenic trioxide).

Arsenic absorbed from GIT , Respiratory system and skin .it transported to liver and excreted in bile and urine .

Used as herbicides and fungicides , defoliant on cotton , wood preservatives .

Ruminates poisoning when lick plant poisoned with arsenic .

In poultry used as feed additive .

Horses poisoned after eat grass clipping from lawns treated with arsenic .

Mechanism of toxic action:-

Arsenic act to inactivated a Co- enzyme (lipoic- acid) associated by pyruvate dehydrogenase.

Uncouple of oxidative phosphorylation.

Has peripheral vasodilatory effect that leads to capillary fluid loss, edema and shock.

Signs of toxicity:-

In acute toxicity: - in oral exposure: - abdominal pain duo to necrotic of GIT, vomiting , watery diarrhea , nausea , weakness, tacky cardia , body temperature, prostration and death .

Sub-acute toxicity: - intoxication in dogs renal and liver damage (icterus, bilirubin urea) GIT damage

Dermal exposure: - blistering, bleeding, secondary infection of skin, cracking.

Diagnosis :-

1- Case history

2- Clinical signs

3- Determination of arsenic level in tissues > 1ppm.

4- Arsenic can be detected in urine for 3 days.

Treatment :-

1- BAL gave 3-4 mg / kg I.M. every 4-6 hr.

2- N- Acetyl cysteine (140- 280 mg /kg/ day) then 70-140 mg/kg/day orally in small animals.

3- Na- thiosulphate (40-50 mg/kg) or thioctic acid 50 mg /kg twice daily I.M. in large animals .

4- Gastric lavage and mineral oil.

5- Fluid therapy

6- High quality diets (small amount).

Chlorates (Na- chlorates) salts:-

Na- chlorates used as herbicides. May mixed in feed.

Cattle attracted to foliage treated with Na- chlorate.

Mechanism of toxic action:-

Na- chlorates ingestion causes hemolysis in red blood cells and conversion of HB to methemoglobin (M Hb) .

Hb Na chlorates MHb may causes mucosal surfaces irritation .

Clinical signs of toxicity: - within 1 hr.

1- Hyper salivation

2- Diarrhea

3- Vomiting

4- Hematuria

5- Hb urea

6- Ataxia

7- Cyanosis

8- Prostration and dyspnea.

Lesion:-

1- Blood stained urine.

2- Erosion in stomach and duodenum.

3- Dark brown tissues.

Diagnosis:-

1- Case history

2- Laboratory analysis of plasma, urine to detect chlorate (present of MHb).

Treatment:-

1- Gastric lavage.

2- Intravenous of methylene blue (4mg/kg) for dogs , 10-15 mg/kg for cattle .

3- Blood transfusion to reduced tissue anoxia.

4- Isotonic saline to elimination of chlorate.

5- Vit . C in dogs and cats.

6- Mineral oil containing Na- thiosulphate 1% inhibit further absorption of chlorate in monogastric animals .

Organic herbicides

Organic herbicides :- are plant growth regulators , some of this group are more toxic than other.

1- Bipyridyl compounds or quaternary ammonium herbicides e.g. (diquat , paraquat).

2- Phenoxy acetic and phenoxy butyric compounds e.g. :- (2,4-D(2,4-dichlorophenoxy acetic acid) and (2,4,5, tricholophenoxy acetic acid).

1- Bipyridyl compounds or Dipyridyl compounds :-

The bipyridyl compound are non-volatile desiccant herbicides, act rapidly , are inactivated on soil contact and rapidly decompose in light . they produce toxic free radicals, tissues can be irritant after contact e.g. mouth lesion after recent spraying of pastures . skin irritation and corneal opacity occur on external exposure to these chemicals and inhalation is dangerous .

Toxicity of paraquat and diquat :-

Dogs and cattle most poisoned paraquat concentrated in lung tissues up to 10 times than other tissues .

It excreted in milk and urine 90-100% in 48 hr.

Mechanism of toxic action :-

Reduce by nicotinamide – adenine dinucleotide phosphate (NADPH)

Electron transfer occur from paraquat to $O^{-}2$.superoxides which reacts unsaturated lipids of cell membranes to form lipid hydroperoxide .

lipid hydroperoxide are unstable spontaneously decompose to lipid free radicals .

ultimately resulting in membrane destruction by lipid peroxidation .

clinical signs :- Diquat

1- Effect in the GIT anorexia , gastritis , GIT distension sever loss of water into the lumen of GIT.

2- Signs of renal impairment

3- CNS excitement convulsion occur in sever affected

4- Lung lesion are uncommon.

Paraquat :-

Immediate effects include :-

Excitement , convulsion or depression and incoordination , gastroenteritis , anorexia and possibly renal involvement and respiratory difficulty .

Diagnosis :-

1- Case history

2- Clinical signs

3- Laboratory test :- a- radiographic change in lung

b- analysis of urine

c- chemical screening spot test in urine up to 8 hr.

d-Chemical analysis of tissues by spectrophotometry (1ml urine blue –green – colure +ve)

e-Lesion :- bronchodilation , pulmonary congestion hemorrhage necrosis of alveolar epithelium , fibrosis of alveoli , emphysema, hepatic and renal tubular degeneration .

Treatment :-

1- Gastric lavage to elimination and reducing pulmonary damage

2- Gastric lavage consist of 30 % suspension of fulter' s earth or 6-7.5 % suspension of bentomite or activated charcoal

Gastric lavage should be repeated every 2-4hr.

3- Cathartics such as sorbitol or sodium sulphate

4- Forced diuresis may be used for elimination (mannitol or furosemide).

5- Vit. E and C .

6- O2 therapy .

Phenoxy herbicides :-

Absorbed from the GIT and bound to protein . distribution to liver , kidney and excreting by urine .

Mechanism of toxic action :-

They inhibit ribonucleous synthesis – uncouple oxidative phosphorylation and number of hepatic peroxisomes.

In dog effect on mucous membrane causing change in the EMG (electrical muscles graphy) .

Clinical signs :-

Depression , anorexia , weight loss , diarrhea , rumen atony , muscles weakness , tremor , hemorrhage , edema , ascites .

In dogs :- myotonia , ataxia , weakness , vomiting , diarrhea , metabolic acidosis .

Diagnosis :-

1- Case history

2- Clinical signs

3- Lab . analysis :- a- alkaline phosphatase (AP).

b- creatine phosphokinase level (liver , kidney , muscles damage) .

4- Chemical analysis :- renal tissues , forage , urine .

Treatment :-

No specific anti dote.

In oral exposure:-

1- Activated charcoal in ruminate.

2- Supportive therapy to treated diarrhea, rumen atony, high quality diet .

Dermal exposure :-

Wash skin and hair with water and soap .