University of Mosul
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Website:

Lecture title: Organophosphorus compounds and Carbamate insecticides

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Summary:

Organophosphorus (OP) compounds and carbamates act in essentially the same manner therapeutically and toxicologically.

E.g. Organophosphorus: Diazinon, Dichlorvos, Malathion

E.g. Carbamate: Bendiocarb Methyl, Propoxur.

Epidemiology

*Occurrence: All animal species are affected. OP compound and carbamate poisoning.

*Source of Toxin

• Grazing in recently sprayed areas, particularly orchards

•Spray used on cereal crops and in orchards carried by wind onto pasture fields

• Hay or cubes made from plants sprayed with organophosphate compounds

• Use of old insecticide containers as feeding utensils

• Too high a concentration of the insecticide in a spray

***Risk Factors**

•Stressed, water-deprived, and chilled animals; the increased susceptibility caused by restriction of water intake is noted especially after oral treatment to control warble fly infestations.

Pathogenesis

*OP compounds are highly toxic and readily absorbed by ingestion, inhalation, and by percutaneous and perconjunctival absorption.

*There are two forms of toxicity: 1- cholinesterase inactivation

2- OP-induced, delayed neurotoxicity.

*The inactivation of cholinesterase by these OP compounds is associated with an increase in acetylcholine in tissues and increased activity of the parasympathetic nervous system and of the postganglionic cholinergic nerves of the sympathetic nervous system.

*The muscarinic effects of acetylcholine are the visceral responses of the

1- Respiratory system and include :marked respiratory distress , an increase in total pulmonary resistance, bronchial constriction and increased mucous secretion .

2- Alimentary tract there is increased peristalsis and salivation.

3- Hypotension and Bradycardia, pupillary constriction, sweating, and abortion.

*The nicotinic effects are the skeletal muscle responses of twitching, tremor and tetany, convulsions, opisthotonos, weakness, and flaccid paralysis.

*Organophosphorus-Induced Delayed Neurotoxicity :This form of toxicity is manifested by distal axonopathy commencing 1 or 2 weeks after the poisoning incident causing regional flaccid paralysis.

Clinical findings

Acute Poisoning

*signs of acute toxicity in animals may occur within minutes of inhalation or ingestion and deaths 2 to 5 minutes later.

Cattle, Sheep, and Goats

1- Salivation, lacrimation, restlessness, nasal discharge, cough, dyspnea, diarrhea, frequent urination, and muscle stiffness with staggering. Grunting dyspnea is the most obvious, often audible from some distance because of the number affected.

2-Protrusion of the tongue, constriction of the pupils, bloat, collapse, and death with or without convulsions or severe respiratory distress.

*Delayed Neurotoxicity

• the signs do not appear for at least 8 days and up to 90 days after the poisoning. Signs include posterior incoordination and paralysis.

Horses

•abdominal pain •muscle tremors, ataxia, circling, weakness, and dyspnea.

Clinical pathology

The estimation of cholinesterase in body tissues and fluids

Necropsy findings

There are no gross or histologic lesions

Differential diagnosis

Cattle

• nicotine poisoning • acute bovine pulmonary emphysema and edema (fog

fever)

Horses • Lead toxicosis

Treatment

Ruminants

1-Atropine sulfate (0.5 mg/kg BW with 1/ 4 given IV and the remainder IM or SC; repeat every 3–4 hours for 1–2 days) (R1)

2- Pralidoxime chloride (2-PAM) (25–50 mg/kg BW IV as a 20% solution over 6 minutes.

Horses

1- Atropine sulfate (0.02 to 0.2 mg/kg BW IV to effect; repeat judiciously SC every 1.5–2 hours)

2- Pralidoxime chloride (2-PAM) (20 mg/kg BW IV; repeat every 4–6 hours as needed).

CONTROL

Most outbreaks occur after accidental access to compounds**.**