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A brief overview on organochlorine insecticide poisoning in animals

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Organochlorines, commonly referred to as chlorinated hydrocarbons, represent the initial generation of insecticides. Primarily employed as contact insecticides and ectoparasiticides, these compounds find extensive application in controlling insects and external parasites.

Sources of poisoning

Animals can be exposed to organochlorines through the ingestion of feeds and water contaminated with these compounds. Additionally, exposure can occur through inhalation or skin absorption during the topical application of organochlorines as ectoparasiticides.

Mechanism of toxicity

Neurotoxic substances, such as chlorinated hydrocarbons, are known to impede proper neural functioning. These compounds possess a high affinity for lipid molecules, enabling them to readily traverse neural membranes and interfere with the normal operation of sodium channels. DDT, for instance, exerts its effects by disrupting potassium transport, impeding the closure of sodium channels, inhibiting ATPases responsible for maintaining sodium-potassium and calcium-magnesium balance, and obstructing the binding of calmodulin to calcium ions involved in neurotransmitter release, among other mechanisms. Cyclodiene compounds, on the other hand, interfere with the Ca2+-Mg2+ ATPase as well as the gamma-aminobutyric acid (GABA) receptors in chloride ion (Cl-) channels, thereby affecting the transit of chloride ions.

The precise contribution of these mechanisms—GABA receptor blockade and Ca2+-Mg2+ ATPase inhibition—to the neurotoxicity of chlorinated cyclodiene compounds remains uncertain. Additionally, an isomer of hexachlorocyclohexane (HCH) known as lindane interacts with GABA receptors.

Clinical symptoms

Dichlorodiphenylethane (DDE) poisoning initially stimulates the central nervous system (CNS), but subsequently leads to depression and ultimately respiratory failure, resulting in death. In cases of chronic poisoning, liver damage, hypoglycemia, depletion of liver glycogen stores, elevated blood lactate levels, and hyperkalemia may occur. Similarly, cyclodiene compound poisoning shares similar symptoms with DDT poisoning, but tends to be more severe. These symptoms encompass teeth grinding, respiratory difficulties, frequent urination, and cracking of the eyelids. Additionally, affected individuals may exhibit peculiar behaviors such as walking backwards, climbing walls, aimless bouncing, and engaging in violent and irrational actions.

Behavioural changes

Initial anxiety, madness syndromes, abnormal posturing, aggressiveness, wall climbing, jumping over unseen objects

1. Neurological symptoms

Increased susceptibility to external stimuli, jaw champing, spasms, and twitching of the muscles in the fore and hind quarters, elevated body temperature, as well as facial and eyelid manifestations can be observed. . Muscle fasciculations and twitches are observed, and if death does not occur during this stage, animals may enter a state of coma.

2. Cholinergic manifestations: Mydriasis, marked salivation, vomiting, micturition and Diarrhoea.

Post-mortem lesions: No specific lesions are observed in the nervous system. However, acute aldrin overdose can lead to hepatitis and severe tubular nephrosis. Chronic toxicoses of DDT and methoxychlor can result in centrilobular necrosis of the liver.

Diagnosis: Organochlorine pesticide intoxication can be diagnosed based on the following criteria:

I. History:

II. Clinical signs: Observing specific symptoms and signs associated with organochlorine pesticide poisoning in affected animals.

III. Post-mortem findings: Identifying characteristic pathological changes during the examination of deceased animals.

IV. Analysis of samples: Testing for the presence of organochlorine chemicals in feeds and various biological samples, including the liver, kidneys, blood, and milk, to confirm exposure and intoxication.

Differential diagnosis: I. Lack of hyperthermia and a history of salt poisoning indicate a different cause.

II. Strychnine poisoning is characterized by tonic convulsions without any abnormalities or changes in locomotion.

III. Convulsions caused by fluoroacetate poisoning are not triggered by external stimuli.

IV. Nicotine intoxication manifests solely with cholinergic symptoms.

V. Anticholinesterase insecticide intoxication presents only with parasympathetic symptoms, without hyperthermia or behavioral changes.

VI. Lead poisoning does not cause any unusual postures.

Treatment: No specific antidote is currently available for organochlorine poisoning. Treatment mainly involves symptomatic and supportive measures, along with the immediate elimination of the source of poisoning.

I. Non-oily purgatives are administered to facilitate the elimination of the toxic compounds. II. In dogs and cats, convulsions can be controlled with the administration of barbiturates (such as pentobarbital sodium) or benzodiazepines. In ruminants, chloral hydrate or pentobarbital sodium may be used for this purpose.