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Abuse of Chemical Substances Cause Poisoning in Dogs and Cats: A Review

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Abstract

Chemical substances are used worldwide as rodenticides, insecticides and herbicides, and also to protect animals against ectoparasites. Poisoning from chemical substances can occur in dogs and cats from both non-accidental and accidental causes. Non-accidental causes include the intentional abuse of chemical substances. This is regarded as animal cruelty and is illegal in many countries. By contrast, accidental causes result from animals ingesting poisonous substances unintentionally such as baits containing rodenticides or eating dead or alive poisoned rodents. Knowledgeable use of poisonous chemicals is vital for the safety of pets. Clinical signs of poisoning in dogs and cats depend on the type of chemical substances, species, lethal dose (LD_{50}) and duration after exposure. This review summarises the common chemical substances that can endanger dogs and cats including rodenticides, insecticides, molluscicides, herbicides, paracetamol and antifreeze chemicals (ethylene glycol).

Key words: Chemical substance, Poison, LD_{50}, Clinical signs, Dogs, Cat

Introduction

Poisoning agents can exist in solid, liquid or gaseous forms.[1] When toxic substances enter the body they cause disturbances in the various processes of organ functions.[2] Classification of toxic substances is based on several criteria such as chemical structure, physical characteristics, the severity of the poison, the target organ causing toxicity and source of the toxic substances (natural or synthetic).[3] Toxicity can be divided into three types as (1) acute toxicity, animals show clinical signs after poisoning once or multiple times within 24 hours, (2) subchronic toxicity, animals show toxicity after consecutive exposure to a small amount of a toxic substance for about 1-3 months and (3) chronic toxicity, animals show toxicity in several organ systems after receiving a small amount of toxic substances for more than three months until toxicity develops.[4]

Dogs and cats are often reported with toxic chemical exposure.[5] Types of toxic chemical substances in pets vary depending on each area. In some areas anticoagulant rodenticides are common, while organophosphates may be found in others.[2, 5] This review addresses toxic chemical substances often found in dogs and cats including rodenticides, insecticides, molluscicides, herbicides, paracetamol and antifreeze chemicals (ethylene glycol).

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Anticoagulant rodenticides

Anticoagulant rodenticide classification follows two groups of chemical structure as hydroxycoumarine and indandione. Hydroxycoumarine subdivides into first-generation (warfarin, coumachlor and coumafuryl) and second-generation (bromadiolone, brodifacoum and flocoumafen), while the indandione group comprises pindone, chlorophacinone and diphacinone.[6,7] The mechanism of anticoagulant rodenticides involves impeding the recycling of active vitamin K1 by inhibiting vitamin K epoxide reductase, which discourages the activation of blood clotting factors (II, VII, IX and X), resulting in blood coagulopathy.[8] Dogs and cats can be poisoned by ingestion of baits containing anticoagulant rodenticide substances or ingestion of dead or alive poisoned rodents.[9] Substance quantity that causes toxicity depends on the anticoagulant rodenticide type. Warfarin shows oral toxicity (LD_{50}) in dogs at 11-323 mg/kg, with 20-50 mg/kg in cats.[7] Clinical signs of toxicity in dogs and cats depend on the amount and duration after ingestion and include anorexia, weakness, pale mucous membrane, urination, polydipsia, hematochezia, melena, hemorrhage on the skin and mucous membrane, nasal bleeding, pulmonary edema, intrapulmonary and pleural hemorrhage.[2,5] Recommended treatment for dogs and cats poisoned during the first 4 hours is ingestion of activated charcoal and emetics, while an antidote (vitamin K1) should be administered immediately.[7]

Bromethalin rodenticide

Bromethalin is a highly potent rodenticide that occurs as an odourless crystalline solid that was developed for anticoagulant rodenticide resistance.[10,11] The bromethalin rodenticide mechanism involves neurotoxicity by decreasing oxidative phosphorylation within the mitochondria of the central nervous system. This impacts ATP production, resulting in reducing the amount of energy to maintain the function of the Na/K-ATPase pump. As a result, the cells lose the ability to regulate osmotic pressure, causing intracellular sodium accumulation and subsequent cell enlargement from the accumulation of water within the myelin sheath. This promotes vacuolation in the nervous system and reduces the efficiency of nerve impulse, transport.[12] Dogs and cats are intoxicated by ingestion of bait directly or ingestion of carcasses of poisoned rodents.[10] The quantity of the chemical substance that causes oral toxicity (LD_{50}) in dogs is 2.5 mg/kg and 0.45 mg/kg in cats.[13] Clinical signs in dogs and cats depend on the amount ingested and duration after ingestion and include weakness, anorexia, vomiting, paralysis, severe muscle tremors, hyperesthesia, hyperthermia, convulsions, coma and death.[13] There is no specific antidote for bromethalin. Supportive care and preventing gastrointestinal absorption are considered.[14]

Cholecalciferol rodenticide

Cholecalciferol rodenticide or vitamin D3 can be ingested by dogs or cats as baits or as food additions.[15] The mechanism increases renal tubular reabsorption of calcium and decreases parathyroid hormone synthesis. This results in the inability to control calcium and phosphorus levels in the plasma.[16] Oral LD_{50} in dogs is reported as 88 mg/kg, while some dogs show clinical signs when given 0.5 mg/kg.[15] The toxic dose reported in cats is doubtful but reports suggest that cats are more resistant than dogs.[16] Clinical signs depend on the amount of cholecalciferol ingested and the hypercalcemia situation and present as polyuria, polydipsia, lethargy, abdominal pain, hematemesis, melena, vomiting, diarrhea and seizures.[17] Treatment involves decontamination of the chemical substances and adjusting the calcium-phosphorus level balance in the bloodstream. This chemical substance has no known antidote.[15]

Strychnine

Strychnine is an alkaloid substance extracted as a white powder from the seeds of *Strychnosnux-vomica* and *S. ignatii* and used as a rodenticide.[18] The mechanism involves a selective competitive antagonist by inhibiting glycine activity at the glycine receptor, with increased glutamic acid and amino acid levels that act as neurotransmitters involved in stimulating the contraction of muscles in the central nervous system. This results in the skeletal muscles becoming more sensitive to stimulation.[18] Oral toxicities reported in both dogs and cats are 0.5 mg/kg.[19] Clinical signs depend on the amount and duration after ingestion and include ataxia,

muscle stiffness, tachypnea, hypersalivation and seizures.[20] There is no specific antidote for strychnine poisoning. Treatment involves controlling the seizures and maintaining oxygen levels within the body to prevent hypoxia and dehydration.[20]

Zinc/aluminium phosphide

Zinc/aluminium phosphide is a powdered rodenticide that smells like acetylene and occurs in many colors such as grey-black, grey-yellow and brown.[21] The toxicity mechanism of zinc phosphide occurs when the chemical substance hydrolyses in humid or acidic conditions to form phosphine, while aluminium phosphide is hydrolysed at a neutral pH. Phosphine gas irritates the gastrointestinal tract and the respiratory system, inducing free radical and oxidative stress that directly affect cell destruction.[22] The toxic dose in dogs and cats is 20-40 mg/kg. Many factors are related to poisoning including the amount of food in the stomach. Dogs and cats with empty stomachs are more resistant to toxic effects. Toxicity increases when ingested with food because the gastric juices catalyse the hydrolysis reaction and convert phosphide to phosphine.[22] Clinical signs in dogs and cats after ingestion include anorexia, vomiting, hematemesis, melena, cyanosis, ataxia and seizures.[21] Supportive care and preventing gastrointestinal absorption are treatment considerations.[6]

Organophosphate and Carbamate

Organophosphate and carbamate are organic phosphorus compounds that are used as ingredients in various insecticides and pesticides. Contained substances include malathion, parathion, diazinon, fenthion, dichlorvos, chlorpyrifos and ethion.[23] Organophosphate and carbamate have similar structural formulae, properties and mechanisms of action.[24] The mechanism works by inhibiting the enzyme acetylcholine esterase, resulting in overstimulation of the muscarinic and nicotinic receptors.[25] LD_{50} in dogs and cats depends on chemical substance types. For marathion, oral toxicity is 500 mg/kg.[26] Clinical signs in dogs and cats of organophosphate and carbamate poisoning depend on the quantity of the insecticide and include hypersalivation, vomiting, depression, dyspnea, ataxia, weakness of hind limbs, bradycardia and seizures.[5, 27] Pralidoxime and atropine are used as antidotes in poisoned pets following supportive care to prevent gastrointestinal absorption.[27]

Organochlorine

Organochlorine insecticides are a group of synthetic chemicals comprising hydrogen, carbon and chlorine.[28] They can accumulate in the environment for a long time with high risk of contamination in the food chain.[28] Organochlorines can be divided according to structural formulations into three groups. The first group contains diphenyl aliphatic compounds such as DDT, methoxychlor, perthane and dicofol. The second group comprises hexachlorocyclohexane and its derivatives such as benzene hexachloride, lindane, gamma hexachlorocyclohexane and paradichlorobenzene, while the third group consists of cyclodienes such as aldrin, dieldrin, endrin, chlordane, heptachlor, endosulfan and isobenzan.[29] The mechanism of toxicity involves stimulation of the central nervous system by disturbing the sodium channel in the neuron membrane. This increases sodium content in the cells and reduces or inhibits potassium exit from the cells, resulting in increased intracellular positive values. Nerves become depolarised, causing pets to have seizures.[29] Dogs and cats are intoxicated through ingestion, inhalation or direct contact. The toxins are well absorbed by ingestion.[8] LD_{50} in dogs and cats depends on the type of chemical substance. Aldrin has oral toxicity of 65-95 mg/kg for dogs and 10-15 mg/kg for cats.[30] Clinical signs in dogs and cats depend on quantity and duration after poisoning and include ataxia, hypersalivation, vomiting and seizures.[28] Supportive care and preventing gastrointestinal absorption are treatment considerations.[28]

Metaldehyde

Metaldehyde is a molluscicide that is used to control snails and scallops in agriculture. Some products such as carbaryl are mixed with insecticides.[31] The action mechanism of metaldehyde is unclear but it has been reported to impact γ-aminobutyric acid (GABA) that is involved in CNS transmission. The reduction of GABA decreases other metabolites such as serotonin and norepinephrine, which are associated with a decrease in the seizure threshold.[32] The oral toxic dose in dogs and cats is 100 mg/kg and 207 mg/kg respectively.[33] After ingestion, clinical

signs in dogs and cats may appear as hypersalivation, vomiting, diarrhea, ataxia, anxiety, tachycardia, tremors and seizures.[32] No specific antidote for metaldehyde toxicosis is available and treatment focuses on preventing substance absorption, with supportive care to control the clinical signs.[31]

Paraquat

Paraquat is a quaternary nitrogen compound that has been used as a herbicide since 1962 in England and later spread worldwide.[34, 35] The toxicity mechanism occurs when the substance is reduced by nicotinamide adenine dinucleotide phosphate (NADPH) in cells to a substance capable of converting free oxygen molecules into superoxide radicals. Superoxide is the primary oxygen free radical produced in mitochondria that leads to cell death.[36] The oral toxic dose in cats is 35-50 mg/kg. Oral LD_{50} in dogs is unknown but higher than for cats.[37] After intoxication, clinical signs in dogs and cats may appear as vomiting, hematochezia, melena, tachypnea, hyperthermia, and hemorrhage at the mucous membrane.[38] Preventive gastrointestinal absorption and supportive care are essential treatments because there is no specific antidote for this substance.[38]

Acetaminophen

Acetaminophen (paracetamol) is a non-steroidal anti-inflammatory drug (NSAID) and most commonly used in humans for mild pain and fever.[39] Cats are susceptible to this drug because they lack methemoglobin reductase enzyme and their red blood cell structure contains up to eight sulfhydryl groups bound to this toxin.[39] Free radicals are produced after the drug's metabolites produce glutathione and appear as oxidative injury. The free radicals cause hemolysis and result in methemoglobinemia.[40] The oral toxicity dose in cats is less than 60 mg/kg, while for dogs it is over 300 mg/kg.[39] Clinical signs of intoxication in dogs and cats appear as facial swelling, hypersalivation, cyanosis and vomiting.[41, 42] Treatment is by administering *N*-acetylcysteine (NAC) to prevent hepatotoxicity and supportive care follows clinical signs.[42, 43]

Ethylene glycol

Ethylene glycol is found in commercial antifreeze for vehicle cooling systems at approximately 95%. This substance is inexpensive and easy to find.[44] After ingestion, oxalic acid from the metabolic process leads to acidosis in the body. Oxalic acid binds with calcium in the serum resulting in the formation of calcium oxalate crystals. These crystals damage the ureter and induce renal failure. Cats are more susceptible to this toxin than dogs; The lethal dose of undiluted ethylene glycol is 1.5 ml/kg in cats and 6.6 ml/kg in dogs.[45,46] After ingestion, clinical signs in dogs and cats appear as vomiting, polydipsia and polyuria, abdominal pain, ataxia, oliguria and seizures.[47] Treatment in dogs and cats aims to decrease the absorption of ingested ethylene glycol and supportive care follows clinical signs.[44]

Pyrethrin-Pyrethroid

The pyrethrin group is extracted from chrysanthemum flowers, while the pyrethroid group comprises synthetic compounds.[48] Both groups have similar properties but the pyrethroid group has higher stability and is not easily decomposed.[49] The pyrethrin and pyrethroid groups comprise many substances used as drugs including cinerin, jasmolin, permethrin, cyfluthrin, cyhalothrin and cypermethrin.[48] The mechanisms of the two groups interfere with neuronal voltage-sensitive sodium channels by inducing slow sodium channel closure. This decreases the intracellular voltage potential, resulting in cellular hyperexcitability.[48] These toxins are used in external forms and absorption of toxin through the skin rarely occurs.[49] The substance mainly enters the body through ingestion and inhalation.[5] The toxic dose in dogs and cats depends on pyrethrin-pyrethroid type. Commercial flea and tick spray products containing 0.09% fenvalerate and 9.0% diethyl-toluamide (DEET) have oral toxic dose at 4 mg/kg in dogs and 2 mg/kg in cats.[49] Clinical signs in dogs and cats from intoxication are mild (anorexia, vomiting), moderate (hypersalivation, blurred vision) and severe (seizures, coma) depending on the pyrethrin-pyrethroid type, quantity and duration after intoxication.[50] Supportive care follows clinical

signs, and decontamination of toxins is the current treatment in dogs and cats.[48]

Macrocyclic lactone

Macrocyclic lactone is a chemical substance widely used to control and eliminate internal and external parasites in pets and livestock.[51] It contains several drugs including doramectin, eprinomectin, ivermectin, milbemycin, moxidectin and selamectin.[52] Macrocyclic lactone exerts its toxic actions through high affinity for ligand-gated chloride channels, particularly those mediated through the neurotransmitters GABA and glutamate. The binding of macrocyclic lactone to glutamate-gated chloride channels causes increased chloride conductance through the cell membrane, resulting in hyperpolarization.[53] LD_{50} in dogs and cats depends on macrocyclic lactone type and status of the *p*-glycoprotein transport system (P-GP) individual. The P-GP is a protein coded by the ABCB1(formerly MDR1), while normal P-GP is tolerant to the toxin. Members in macrocyclic lactone such as ivermectin gave oral toxicity in cats at 1 mg/kg, dogs 80 µg/kg (ABCB1 defective) and 0.2-1 mg/kg (ABCB1 normal).[53] Clinical signs of poisoning with these substances are related to the nervous system. In cases of acute toxicity from high doses, exposure can be induced within hours and pets show signs of depression, ataxia and hypersalivation. In severe cases, dogs and cats may fall asleep and become unconscious.[54] No antidote for this substance exists. Supportive care is an important treatment.

Fipronil

Fipronil is a broad use chemical insecticide. This substance is used in veterinary medicine to prevent and eliminate external parasites such as ticks, fleas or mites.[55] The mechanism of toxicity inhibition at the GABA receptor-chloride complex causes sustained activation of the central nervous system, resulting in convulsions, loss of consciousness and death. Fipronil has a selective binding that only catches glutamate-gated GABA chloride channel, which is found only in invertebrates. Thus, invertebrates are the group most at risk of being affected.[55] Based on toxicity in dogs and cats, fipronil is classified as a moderately toxic substance. Skin contact alone produces little or no toxicity. Oral LD_{50} in dogs and cats is necessary receipt accumulate several months will show clinical signs (0.2-0.5 mg/kg/day).[56] Clinical signs are often neurological-related symptoms including loss of appetite, muscle tremors, and aggressive behavior.[57] There is no specific antidote for the toxicity of fipronil. Supportive care follows clinical signs and decontamination is a treatment consideration.[55]

Amitraz

Amitraz is an insecticide used to prevent and eliminate insects such as ticks, fleas, and mites in pets and livestock.[58] The product can be a powder, collar, spray or bath. The mechanism of action interferes with the function of the nervous system This results in paralysis of the insect's mouth, with inability to suck blood from the host. High doses of amitraz can cause toxicity to mammals through action of the alpha 2-adrenergic agonist and a monoamine oxidase inhibitor (MAOI) that influence the nervous system and cause cardiovascular system disorder.[58, 59] Oral LD_{50} in dogs is 100 mg/kg but less in cats.[30] The toxicity in dogs and cats is divided into two main categories as acute toxicity and chronic toxicity. Acute toxicity usually appears within 30 minutes to 2 hours after intoxication. Clinical signs in acute intoxication include nausea, vomiting, diarrhea, arrhythmia, bradycardia, decreased blood pressure and hypothermia. Chronic intoxication affects the endocrine and reproductive systems.[58, 60] There is no specific antidote for amitraz poisoning. Treatment should prevent substance absorption, with supportive care to control the clinical signs.

Conclusions

Toxic poisoning in dogs and cats can occur as a result of non-accidental and accidental causes. Dogs or cats can accidentally ingest baits containing rodenticide substances, or dead or alive poisoned rodents if the owner lacks knowledge of the correct use of the chemicals. Non-accidental causes include intentional abuse, which is considered animal cruelty and is illegal in many countries. Clinical signs in dogs and cats after intoxication depend on the type of substances, quantity ingested and duration after exposure. Defining the type of substance requires variation assessment including information from the owner, physical evidence, clinical examination on

pets and detection of substances in the laboratory. Treatment should be managed immediately when known pets are exposed to toxic substances to avoid progress of the clinical signs.

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