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Common herbicide and acaricide toxicities in animals- a review

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Abstract

Herbicides are phytotoxic chemicals that are routinely used for unwanted plants and weed control. Most toxicity problems in ruminants result from exposure to excessive quantities of herbicide because of improper or careless use or disposal of containers. Herbicides vary widely in their composition, toxicity, associated toxicity, mechanism of action, and use. Acaricides are pesticides used to kill ticks and mites. Poisoning by acaricides are mainly caused by direct application, by ingestion of contaminated feed or forage, or by accidental exposure.

Key words: herbicides, phytotoxic, acaricides, ticks and mites, accidental exposure

Introduction

Herbicides or weed killers are phytotoxic chemicals used for destroying various weeds or inhibiting their growth (Gupta, 2018), considered as a revolutionary tool in agricultural practices. The consumption of herbicides in developing countries is low because weed control is mainly done by hand weeding (Gupta, 2007). Herbicides forms 48 percent of total pesticide usage andmost of intoxications occur due to improper use or careless disposal of containers. Acaricides are the chemical agents used to kill ticks and mites, which include carbamates, organochlorides, organophosphates, formamides etc. Poisoning by acaricides are mainly caused by direct application, by ingestion of contaminated feed or forage, or by accidental exposure. Important herbicide and acaricide poisoning in livestock are discussed here.

Herbicide toxicities in livestock

1.bipyridyl compounds:

Poisoning of Bipyridyl compounds occur in animals *via* ingestion, inhalation and dermal exposure. Paraquat and Diquat are two important poisonous herbicides in this group, of which paraquat is more toxic. Bipyridyl compounds are absorbed rapidly and incompletely from the gastrointestinal tract. Paraquat is commercially formulated as dichloride salt, but it is least selective and more toxic, it is no more commercialized in developed countries (Constable *et al.*, 2017). Toxicity is more in cattle and sheep.

Mechanism of action of bipyridyl compounds involves cyclic reduction – oxidation reactions, which results in the formation of reactive oxygen free radicals and deletion of Nicotinamide Adenine dinucleotide phosphate. Unstable singlet oxygen activates lipids in cell membrane and results in lipid peroxidation of cell membranes, membrane dysfunction and cell death (Gupta, 2018). But their target organ varies, paraquat mainly accumulate in the lung tissue and produce acute alveolitis. Gastrointestinal tract, kidneys and the liver form the targets of diquat (Hayes, 1982).

Moderate to severe toxicity occurs in patients who ingest more than 40-80 mg/kg and the poisoned animals may die from pulmonary haemorrhage and fibrosis. Clinical manifestations comprise of 3 phases, initial phase of corrosive effects of herbicide, renal failure and hepatocellular necrosis during second phase and third phase is the pulmonary fibrosis with poor prognosis (Dinis Oliveira *et al.*, 2008). Gross lesions in paraquat poisoning comprise lung congestion, edema and pulmonary hemorrhage. There may be atelectasis and fibrosis (Gupta, 2018).

Oral ingestion of Diquat produce vomiting, abdominal cramps, diarrhea and erosion of GI mucosa (Vanholder*et al.*, 1981). The therapy of bipyridyl compounds focused on 3 important aspects, prevention of toxin absorption from the GI tract, speed up excretion of drug from the body and therapy focused against the mechanism of action (Lock and Wilks, 2010).

2.carbamates, thiocarbamates and dithiocarbamates:

Members of the group includes herbicides like asulam, barban, chorampham(derivatives of carbamic acid), butylate, diallate, triallate (derivatives of thiocarbamic acid), and metham sodium (Gupta, 2018). Toxicity is not reported at lower concentrations. Repeated oral ingestion produce marked alopecia (Hurt *et al.*, 2010). In cattle, diallate produce anorexia, muscle contractions, exhaustion and prostration. Thiobencarb appears to increase the permeability to blood brain barrier and induce toxic neuropathies (Gupta, 2018).

3.dinitrophenol compounds:

2,4- Dinitrophenols (DNP) and Dinitro-ortho cresols (DNOC) are the important toxicants of this group of herbicides. Poisoning of animals occur *via* ingestion, inhalation or percutaneous absorption when they get access to herbicide treated fields or spill overs of preparations and cause potential hazards. After ingestion, inhalation or percutaneous absorption, toxicant cause uncoupling of mitochondrial oxidative phosphorylation and resulting in marked increase in fat metabolism (Miranda*et al.*, 2006)

Doses of 25-50mg/kg bodyweight (BW) is usually toxic to almost animals. Clinical signs appear within 2-5 days of ingestion which include fever, ataxia, acidosis, muscular weakness, tachycardia, convulsions followed by coma and death within 24-48hrs (Gupta, 2018).

4.phenoxy acid derivatives:

Phenoxy acid derivatives form one of the most extensively used herbicides which include 2,4- dichloro phenoxy acetic acid (2,4 D), 2,4- DB, 2,4,5 T, dalapon and silvex. Among these, 2,4 D is the most extensively used weedicide. Absorption from GI tractis rapid and excreted unchanged *via* urine within 3.5 to 18hours (Gupta, 2018). Rapid absorption occurs at lower dose within a range of 0-4mg/kg/day than at higher dose (Pelletier *et al.*, 1989).

Exact mechanism of action is unknown. It is proposed that they depress ribonucleases and uncouple mitochondrial glycolytic oxidative phosphorylation and increase of liver peroxisomes occur with 2,4 D and 2,4,5 T (Sandhu and Brar, 2000).

Members of this group are potentially nontoxic, neither induce immunotoxicity, neurotoxicity nor have any carcinogenicity or mutagenicity in laboratory animals or dogs (Munro *et al.*, 1992). Oral LD50 for dogs is 25-250 mg/kg/day (Kennepohl*et al.*, 2010). Dogs are more sensitive to the dose of 100-800mg/kg body weight produce nervous signs ataxia, posterior weakness, myotonia, vomiting, diarrhea and acidosis (Gehring *et al.*, 1976). Teratogenicity and reproductive abnormalities are not reported with 2,4 D except at toxic doses above 50mg/kg BW/day (Kennepohl*et al.*, 2010). Oral dosing of 150-188mg/kg BW in adult cows is toxic and cause recumbency, ruminal stasis, salivation, tachycardia. Clinical signs occur in sheep with toxic dose of 10mg/kg (Constable *et al.*, 2017).

Silvex is potentially toxic at small doses and produce ill effects in dogs (Sandhu and Brar, 2000). Repeated doses of 10mg/kg BW for one month causes death in sheep (Constable *et al.*, 2017).

5. Organophosphorus compounds:

Glyphosate and glufosinate are the commonly used OP herbicides. Glyphosate is considered as one of the least hazardous herbicides (Duke *et al.*, 2003). Rate of absorption is low via orally and dermal routes. They do not accumulate in tissues and excreted mainly *via* feces (Gupta, 2018). Amino methyl phosphonic acid (AMPA) is the main metabolite having no toxicological significance (JMPR, 2004). Compounds available as free acids and marketed as trimethyl sulfonium or isopropyl amine salts of glyphosate and ammonium salts of glufosinate.

Mechanism of toxicity occur *via* selectively inhibiting 5-enol pyruvyl shikimate-3-phosphate, an enzyme plays a key role in chorismate pathway for the biosynthesis of aromatic amino acids. This pathway is characteristics in plants, so glyphosate poisoning is selective toxicity to plants (Franz *et al.*, 1997). Acute oral dose is 750mg/kg BW, which is low. Glyphosate neither produce reproductive failures nor carcinogenicity

(Duke and Powles, 2008). Clinical signs in dogs and cats include eye irritation, skin and upper respiratory signs. Gastro intestinal disturbances, staggering and hind leg weakness are reported (Susan, 2003).

Poisoned cats exhibit vomiting, tremors, muscle fasciculations, drowsiness. Dogs are frequently poisoned species and produce increased muscular activity, convulsions, and Gastro-Intestinal signs. No clinical signs developed in exposed horses or goats (Cortinovis*et al*., 2015).

Glufosinate is slightly more hazardous than glyphosate (Gupta, 2018). Acute toxicity produces CNS ill effects and hypothermia in animals (Ebert *et al*., 1990). Ammonium preparations of glufosinate, due to their surfactant induced penetration into CNS are more poisonous (Watanabe and Sano, 1998).

6.Triazines and triazoles:

Members of this group have been extensively used in agriculture in all over the world for many years (Breckenridge *et al*., 2008). There are symmetrical and asymmetrical triazines. Major commercial symmetrical triazines include simazine, atrazine, propazine, cyanazine, ametryn, and prometryn (Breckenridge *et al*., 2010).

Triazines block photosynthetic electron transport and reversibly inhibit photosynthesis in organisms (Trebst, 2008). Herbicides of this group are generally not acutely toxic, and well tolerated when administered to animals over a long duration, and are generally not developmental or reproductive toxin, and are not carcinogenic or mutagenicin experimental animals (Breckenridge *et al*., 2010).Cyanazine is an exception, which is more acutely toxic, weak mutagen and cause developmental toxicity in rats and rabbits in high doses (Hodgson and Meyer, 1997). Sheep and cattle are more susceptible to the acute toxicity and main clinical manifestations include anorexia, haemotoxic, hypothermia, locomotor disturbances, irritability and hypersensitivity (Sandhu and Brar, 2000).

Toxicity occurs in sheep with dose of 30mg/kg for 30-60 days. Grazing in pastures within 1-7 days of treatment cause death in sheep (Gupta, 2018). Atrazine poisoning in sheep is associated with paralysis, grinding of teeth, diarrhoea, tachycardia and in cattle, there will be salivation, tenesmus, shift gait and weakness (Constable *et al*., 2017). Simazine is excreted *via* milk and cause a public health concern (Susan, 2003) and produce tremors, tetany, paraplegia and prancing gait with head held against chest. Colic is noted in poisoned horse (Constable *et al*., 2017).

7.ureas/thioureas:

The commercially available ureas and thioureas include diuron, meturon, isoproturon, linuron, buturon, fenuron, neburon, parafluron and tebuthiuron (Gupta, 2018). With the exception of tebuthiuron, other polyureas are relatively less toxic to animals. They are readily absorbed from the GI tract, and metabolized by dealkalization of urea methyl group (Gupta, 2018). Predominant metabolite in urine is N-(3,4-dihloropheny) urea and excreted unhanged predominantly in urine.

Diuron and monuron are hepatic enzyme inducers. Acute oral LD50 for diuron is 3-4g/kgand produce toxicity signs like drowsiness, ataxia, irritability, and tachypnoea. Animals produce hypothermia, glucosuria, proteinuria, aciduria and significant loss of body weight(Liu J, 2010). The phenyl urea herbicides like linuron and monuron are rodent liver carcinogen (Gupta, 2018). Intensity of toxicity depends on dose and clinical signs disappear by 72 hours after exposure in animals (Boyd and Krupa, 1970).

Toxicity of thimeturon occurs at single oral toxic dose of 4g/kg in a sheep (Mohamed *et al.*, 1993). Repeated high doses produce toxicity signs include depression, salivation, grinding of teeth, chewing movement of jaws, mydriasis, dyspnoea, drowsiness. Serum analysis reveals elevated levels of Alanine aminotransferase, Aspartate aminotransferase, Lactate dehydrogenase and Blood urea nitrogen (Liu J, 2010).

8.substituted anilines:

Atachlor, Acetachlor, Butachlor, Metolachlor and Propachlor are the important commercially available aniline herbicides. Compounds are well absorbed from the GI tract and metabolism occurs in the liver and excreted via urine and feces (Gupta, 2018). Mechanism of toxicity is associated with oncogenic effects produced in nasal turbinates, thyroid glands and stomach (Heydens et al., 2010).

Low doses produce no adverse effects, but long-term exposure produce hepatotoxicity and splenic effects in dogs. Members of these group are slightly hazardous except for butachlor which is relatively nontoxic (Gupta, 2018). They neither produce teratogenicity or reproductive failures.

Metolachlor is associated with incidence of liver tumors in laboratory animals and classified as human carcinogen (Monsanto 1991, Wilson and Takei, 1991). High doses of propachlor produce GI erosion, ulceration and gastric mucosal hyperplasia and liver necrosis. Its adverse effects in dogs include poor palatability and weight loss in dogs. It also produces slight developmental or reproductive effects (Gupta, 2018).

Important acaricide toxicosis:

1.organophosphorus compounds abd carbamates:

Organophosphorus(OP) compounds exert their action by competitively inhibiting cholinesterase (ChE), results in concentration of Acetylcholine at neuromuscular system (Abdelsalam, 1987). Mechanism of action of both OP and carbamates are in same manner, but the binding to cholinesterase enzyme is reversible and degradable with carbamates, potentiates them less hazardous. OP compounds are absorbed by oral ingestion, inhalation, percutaneous and per conjunctival routes. Young animals are more susceptible to toxic effects. Due to high levels of testosterone which renders chlorpyrifos more toxic to males, its use is not recommended in bulls over 8 months of age (Constable *et al.*, 2017).

OP compounds can inhibit enzymes like trypsin, chymotrypsin, and milk lipase and can interfere hepatic metabolism of steroid and facilitates release of adrenal steroids and catecholamines (Abdelsalam, 1987).

Clinical signs of toxicity include anorexia, vomiting, abdominal pain, GI abnormalities, frequent defecation, diarrhea, sweating, excessive salivation, lacrimation, and nasal discharge (Abdelsalam, 1987). Poisoned cattle exhibit dyspnea, ataxia, salivation, lacrimation and tremors (Mitema and Masha, 1984). Pigs exhibits salivation, paresis and paralysis of eyelid. Toxicity signs in dogs include ataxia, muscle fasciculations, vomiting and depression. Death occurs due to asphyxia from acute respiratory failure.

2.formamidine toxicity:

Amitraz and Chlordimeform are two widely used formamidine pesticides. Environmental Protection Agency (EPA) classified amitraz as class III- slightly toxic by oral and inhalation, and as class II- moderately toxic by dermal route, and as class IV nonirritant to skin (Del Pino *et al.*, 2015). Compound is highly soluble and absorbed rapidly through skin and mucous membrane (Chakraborty *et al.*, 2011).

Main mechanism of toxicity occurs via activation of α2 adrenergic receptors as it plays an important role in induction of hypotension, brady cardia, polyuria, GI hypermotility and hyperglycemia (Costa *et al.*, 1989). Amitraz is a histamine-H1 receptor antagonist in pig ileum and produce acute gastrointestinal toxicity (Pass and Seawright, 1982). They also inhibit prostaglandin synthesis and possess antipyretic and anti-inflammatory effects at dose rate of 5-80mg/kg in rats (Yim*et al.*, 1978). They can also inhibit monoamine oxidase (MAO) (Knowles and Roulston, 1972).

It can pass blood brain barrier (JMPR, 1998) and produce neurotoxic effects like mydriasis, vomiting, CNS depression, sedation, loss of righting reflex, motor incoordination, coma and behavioral effects (Florio *et al.*, 1990). Amitraz disrupts sex hormones, insulin, glucagon and PGE2. As mono amines participate in regulation of sexual behavior, ovulation, and implantation, amitraz may have serious effects on fertility and reproduction (Del Pino *et al.*, 2015).

Clinical signs in poisoned dogs include depression and reluctance, polyuria, bradycardia and mydriasis. Low doses of atipamezole effectively reverse clinical signs within 10 minutes of injection. Dose rate of 50µg/kg IM is effective, therapy should be repeated after 3-4 hours if necessary (Hugnet*et al.*, 1996). Decontamination with cathartics and oral administration of activated charcoal found effective immediately after toxicosis. Topical residues are removed using bathing with tepid water and soap (Constable *et al.*, 2017).

3.organochlorine compounds:

Members of this group includes aldrin, dieldrin, lindane, chlordane, endosulfan, heptachlor, toxaphene, methoxychlor and Dichloro Diphenyl Trichloroethane (DDT)

(Raisbeck, 2013). Most of Organochlorine (OC) compounds have an offensive odor and they are less acutely toxic than OP or carbamates for similar purpose.

Adipose tissues serve as sink for OC during prolonged low dose exposure, the adipose OC concentration increases and acute intoxication occurs if the animal is forced to lose weight rapidly and potentially toxic dose become available to general circulation. Enterohepatic recycling serves to prolong the persistence of OC in the body (Raisbeck, 2013). Organochlorines like endosulfan and lindane and OPs were the insecticides more commonly involved in animal poisoning (Berny *et al.*, 2010).

OC insecticides are classified as cumulative poison (Harrison,1971). Organochlorine compounds produce nervous stimulation and hypersensitivity and they exhibit tremor and convulsions (Raisbeck, 2013). Cats are more sensitive to OC compound poisoning (Ensley, 2012). Most OCs interfere with axonal transmission of impulses and therefore disrupt nervous system functions. DDT inhibits mechanism in which sodium influx is stopped and potassium outflux is started during an action potential, there by rendering fiber hyperactive (Doherty, 1984). Compounds like dieldrin, aldrin apparently enhance Neurotransmitter release at synapse and increase brain ammonia concentration by impairing glutamine synthesis (Omer, 1971).

Clinical signs are exhibited within few minutes to 1-2 days after exposure of toxic dose and exhibit incoordination and hypersensitivity, head and neck muscle fasciculations, abnormal posture, continuous chewing movements, finally tonic-clonic convulsions and progress to coma and death. Other signs include weakness, vomiting and respiratory depression. Gross lesions include hepatocellular degeneration and necrosis, renal tubular degeneration with OC compounds (Raisbeck, 2013). There are no specific antidotes for OC intoxication. There are only symptomatic and supportives on preventing further adsorption of toxicant (Raisbeck*et al.*, 1989). Prognosis is guarded to good depending on dose exposed and initiation of treatment (Raisbeck, 2013). Sedate the animal with anesthetics or muscle relaxation to minimize convulsions. Supplemental oxygen may be given through an intranasal tube or oxygen cage or via tracheal intubation. Provide parenteral nutrition. If oral exposure is suspected, provide adsorbent such as activated charcoal or cholestyramine and saline cathartics. Wash the animal with soap and water if dermal exposure is suspected.

Poisoning can be differentially diagnosed from infectious encephalitis, lead poisoning, rabies, eclampsia, canine distemper, and convulsant poisons like OP and carbamates. Sheep decontaminate more quickly than cattle and animals on high plane of nutrition also eliminate toxins more quickly (Constable *et al.*, 2017).

4. Macrocyclic lactones toxicosis:

Macrocyclic lactones (ML) are commonly used as insecticide, acaricide and nematicide in various animal species and members include doramectin, eprinomectin, iveremectin, milbemycin, moxidectin and selamectin. Ivermectin, a semi synthetic ML is one of the most widely recognized one because of their efficacy and wide safety margin (Gwaltney-Brant *et al.*, 2018). Ivermectin is approved for use in ruminants, swine and

horse at dosage of 0.2 – 0.5mg/kg orally, topically or subcutaneously and is not approved for lactating animals. Selamectin, novel semisynthetic avermectin is indicated in ear mite infestation in dogs and cats and to control sarcoptes mange in puppies and in treatment of round worm (*Toxocaracati*) in kitten and hookworm (*Ancylostoma caninum*) in puppies. Topical application of selamectin at dosage of 6mg/kg has a broad spectrum of activity against many endectoparasites in dogs and cats (Dryden *et al*., 2001). As the ML are very safe, toxicosis generally occurs due to accidental overdosing or when the species-specific products are misused among different animal species (Merola and Eubig, 2012). MLs have prolonged antiparasitic activity due to slow absorption, wide tissue distribution and low rate of metabolism and slow excretion (Lanusse *et al*., 2009). After topical administration, selamectin undergoes a multiphasic distribution, it is absorbed into blood stream and excreted via bile into GI tract and undergoes reabsorption and selectively distributed to sebaceous glands of skin (Gupta *et al*., 2005) and it is widely used to control hematogenous, GI and integumentary parasites.

MLs cannot pass blood brain barrier (BBB) through the action of a 170kDa transmembrane protein, P- Glycoprotein system (P-GP) which limit systemic and brain uptake of MLs, and it is coded by ABCB1, former MDR1 gene located mainly in intestine, liver, kidney and brain (Mosher and Court, 2010).

Mechanism of toxicity is exerted by binding to GABA and glutamate gated chloride channels. Glutamate channels are found uniquely in nematodes and arthropods (Gwaltney-Brant *et al*., 2018) and binding to them cause increased chloride conductance through cell membrane, resulting in hyperpolarization and flaccid paralysis of parasites (Lanusse *et al*., 2009). Toxicosis occurs when MLs access to brain when the P-GP is overwhelmed during overdosage, or when it became defective or compromised (Gwaltney-Brant *et al*., 2018).

General signs in ML toxicosis in almost all species are those of CNS depression, although in some species, GABA mediated cholinergic effects have been reported. Ocular effects with ML overdose include mydriasis, miosis, absent menace response and blindness and in all reported cases, vision returned on recovery (Gwaltney-Brant *et al*., 2018). In affected dogs, signs include hypersalivation, vomiting, lethargy, ataxia, tremors, hyperthermia or hypothermia, disorientation, head pressing, seizures, coma and death and in cats, mild diarrhea, posterior ataxia, myosis or mydriasis, vocalization, ataxia, tremors, sternal recumbency, coma and death (Merola and Eubig, 2012). Toxicity signs in calves include depression, ataxia, diarrhea, dyspnea, tachycardia, recumbency, increased respiratory rate, muscular fasciculations, mydriasis, extensor rigidity of limbs (Gupta, 2007). Affected horse may exhibit depression, ataxia, mydriasis, decreased respiratory rate and drooping of lower lip (Leaning, 1983).

Diagnosis is performed based on the history of exposure to the product, clinical signs and residue analysis using body tissue or fluids. GI content, liver, fat and feces are analyzed for presence of residue and brain tissue could be utilized for confirmatory diagnosis.

Treatment for intoxication is symptomatic and supportive which include limiting the systemic absorption of toxicant and monitoring for possible clinical effects and management of existing signs as there is no specific antidote for intoxication. Activated charcoal should be administered orally if the animal is stable, and to counteract enterohepatic recycling, multiple doses are advised and it is the valuable decontamination option when overdosage by ingestion occurs (Gwaltney-Brant *et al*., 2018). Surgical excision of injection site can be considered as a treatment option when there is a life threatening toxicosis occur via subcutaneous injection and bleb is palpable (Beasley *et al*., 1999). Emesis may be effective if it performed within 2 hours after oral ingestion of ML (Mealey, 2006).

The patient should be monitored for the development of CNS signs. Recumbent or comatose animal require good nursing care include thermoregulation, soft bedding and frequent turning of animal to prevent ulceration (Mealey, 2006).

Infusion of intravenous Lipid Emulsion (ILE), a novel approach has been indicated in ML toxicosis in dogs, cats and horses (Pollio *et al*., 2016). The exact mechanism by which ILE is effective in toxicosis is unknown, but ILE seem to be the most effective in treating overdosage of lipid soluble medications expands the amount of plasma lipids, which acts as vascular lipid sink which pulls drugs from CNS into systemic circulation and facilitates its metabolism and excretion (Bruenisholz*et al*., 2012) and it should be administered as 20% solution, give an initial bolus of 1.5ml/kg slow and then 0.25ml/kg/min as CRI for 30-60 minutes. The treatment should be repeated after 4 hrs. of CRI, if there is no lipemia in serum. If hyperlipemia or orange or yellow colored serum noticed, check the serum for resolution in every 2 hours. Treatment should be repeated after resolution of lipemia in serum. Do not give the treatment for more than 3 times, if there is no clinical response (Gwaltney -Brant *et al*., 2018).

Side effects of ILE includes pancreatitis, fat emboli formation, immunosuppression, phlebitis, thrombosis and hepatic lipidosis (O'Brien *et al*., 2010).

5. Pyrethroid toxicity:

Pyrethrins are natural insecticides from the extract of pyrethrum flowers (Proudfoot, 2005). As these are dermal applicants, most commonly toxicity occurs *via* dermal route, chance of oral and inhalation exposure occurs due to grooming in animals (Anadon*et al*., 2013). Pyrethrin insecticides have a wide margin of safety when compared to OC or OP insecticides (Ensley, 2018). Pyrethroids are lipophilic and distribute more to adipose tissues, nervous system in addition to liver and kidney as there are high lipoid content (Kim *et al*., 2008). Pyrethroids are rapidly hydrolyzed in GI tract and they are metabolized by mixed function oxidases and esterase (Ensley, 2018). Their mechanism of action is by slow down the opening and closing of sodium channels, resulting in excitation (Marban*et al*., 1989). They also affect the voltage dependent chloride channels found in brain, nerve, muscles, salivary gland and control cell excitability (Ensley, 2018). They produce a phenomenon called knockdown in acarids (Narahashi, 1985) caused by inhibiting cell, but does not cause a lethal effect.

Main signs of intoxication in animals include salivation, vomiting, hyperexcitability, tremor, seizures, dyspnea, weakness, prostration and death (Murphy, 1996). Treatment is symptomatic as there is no specific antidote. As dermal exposure is the main route, wash the animal with mild detergent and water. Avoid use of shampoo that may contain additional insecticides, which may increase the exposure to insecticides (Ensley, 2018). Emetics and gastric lavage are indicated within 1-2 hours of oral ingestion. Activated charcoal and saline cathartics are indicated to reduce the extent of absorption and hasten elimination. Hyperexcitability and seizure are controlled using diazepam and barbiturates. Prognosis of pyrethroid toxicity is usually good due its low toxicity (Ensley, 2018).

Conclusion:

Herbicides and acaricides, routinely used to control noxious plants and ectoparasites are potentially toxic to animals when exposure occurs. Most of these phytochemicals are quite selective for specific plants and have low toxicity for mammals; other less selective compounds (e.g., arsenicals, chlorates and dinitrophenols) are more toxic to animals. Most animal health problems including reproduction, which is affected by endocrine disruption, result from exposure to excessive amounts of herbicides or acaricides because of improper or careless use or disposal of containers. The residue potential for most of these chemicals is comparatively low.

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