Top 15 poisons affecting dogs and cats

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INTRODUCTION

Each year, the **ASPCA Animal Poison Control Center** manages hundreds of thousands of poisoning calls on common toxicants in the house, yard, garden, or garage. This lecture reviews the top 20 poisons affecting dogs and cats, and reviews the mechanism of toxicosis, clinical signs, and overall treatment. In the veterinary poisoned patient, the goal of decontamination is to "inhibit or minimize further toxicant absorption and to promote excretion or elimination of the toxicant from the body."[1,2] When treating the poisoned patient, the clinician should have an understanding of the toxic dose (if available), the pharmacokinetics (including absorption, distribution, metabolism, and excretion), the underlying mechanism of action, and the potential clinical signs that can be observed with the toxicant.[2] This will help determine appropriate decontamination and therapy for the patient. If this information is not readily available, the reader is advised to contact the **ASPCA Animal Poison Control Center** (888-426-4435) for life saving, 24/7 advice as needed. For further review on decontamination and specific treatment, attendees are referred to a veterinary toxicology book for more detailed review.

CALCIUM CHANNEL BLOCKERS, BETA-BLOCKERS, ACE-INHIBITORS, STATINS AND DIURETICS

Certain cardiac medications include broad categories such as calcium channel blockers (CCB), beta-blockers (BB), and angiotensin-converting enzyme (or "ACE") inhibitors. These medications are commonly used in both human and veterinary medicine to treat underlying cardiac disease or hypertension. Each category of cardiac medication has different margins of safety. CCB and BB toxicosis should be treated aggressively, as these two categories of medications have a narrow margin of safety. Toxicosis of these agents can result in myocardial failure, severe bradycardia, and hypotension; untreated, cardiac output becomes reduced, and secondary severe hypoperfusion and acute kidney injury (AKI) can potentially develop.[3-5] With ACE-inhibitors, severe overdoses can cause hypotension, dizziness, weakness, and hypotension. In general, there is a wider margin of safety with ACE-inhibitors, which are typically considered much safer. Pets ingesting small amounts of ACE-inhibitors can potentially be monitored at home, unless they have underlying disease (e.g., kidney failure, cardiac disease, etc.). With ACE-inhibitors, ingestions > 10-20X a therapeutic dose are generally considered toxic, and can result in severe clinical symptoms (e.g., hypotension).[5] Treatment for any cardiac medication includes decontamination (e.g., emesis induction, gastric lavage, activated charcoal (AC) administration), blood pressure monitoring, aggressive IV fluid therapy if hypotension is detected, and blood work monitoring. With severe toxicosis, the use of high-dose insulin therapy or intravenous lipid emulsion may be warranted as a potential antidote for calcium channel blocker toxicosis.[3]

SELECTIVE SEROTONIN RE-UPTAKE INHIBITORS (SSRI)

Selective serotonin re-uptake inhibitors (SSRIs) are a class of medications that are commonly used in human medicine for depression. Common examples include the following drugs:

- Fluoxetine (Prozac® in human beings; Reconcile™ in veterinary medicine)

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- Citalopram (Celexa®)
- Paroxetine (Paxil®)
- Sertraline (Zoloft®)

Other similar drugs include selective norepinephrine re-uptake inhibitors (SNRIs), which include common drugs like duloxetine (Cymbalta®), nefazodone (Serzone®), and venlafaxine (Effexor®). SNRI and SSRI drugs result in similar clinical signs of toxicosis, and therefore are treated the same. In veterinary medicine, SSRIs are used for a wide array of behavioral problems, including feline urine spraying, canine separation anxiety, lick granulomas, etc. These SSRI drugs work by blocking the reuptake of serotonin in the pre-synapse, thereby increasing the levels of serotonin in the pre-synaptic membrane. In small animal patients, common clinical signs from SSRIs include the following:

- Sedation or central nervous system (CNS) stimulation
- Anorexia
- Lethargy
- Serotonin syndrome

Clinical signs of serotonin syndrome include: gastrointestinal (GI) signs (e.g., hypersalivation, vomiting, diarrhea, abdominal pain) and CNS signs (e.g., stimulation, mydriasis, tremors, seizures, hyperthermia secondary to tremoring and seizuring). Treatment for antidepressants includes decontamination (ideally done at a veterinarian, due to the rapid onset of clinical signs), sedation (e.g., with acepromazine or chlorpromazine), intravenous (IV) fluid therapy, blood pressure and electrocardiogram (ECG) monitoring, thermoregulation, muscle relaxants (for tremors; methocarbamol 22-55 mg/kg, IV, PRN), anticonvulsants (e.g., phenobarbital 4-16 mg/kg, IV, PRN; diazepam 0.25-0.5 mg/kg, IV, PRN), serotonin antagonists [e.g., cyproheptadine (1.1 mg/kg for dogs or 2-4 mg *total* per cat) PO or rectally q. 6-8], and supportive and symptomatic care. In general, the prognosis for antidepressant toxicosis is excellent.

AMPHETAMINES

Amphetamines are used for a variety of medical and illicit reasons. Legal forms include prescription medications for attention-deficit disorder/attention deficit-hyperactivity disorder (ADD/ADHD), weight loss, and narcolepsy. Examples of amphetamines include:

- Dextroamphetamine
- Amphetamine (Adderall®)
- D-amphetamine (Dexedrine®)
- Methamphetamine (Desoxyn®)
- Lisdexamfetamine (Vyvanse®)

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Illegal forms of amphetamines include street drugs like methamphetamine, crystal meth, and ecstasy. This class of drugs acts as sympathomimetic agents, meaning they stimulate the sympathetic system. Amphetamines also cause stimulation of α and β-adrenergic receptors, and stimulate release of serotonin and norepinephrine; this results in increased catecholamine stimulation in the synapse. Amphetamines also increase release of serotonin from the presynaptic membrane, resulting in serotonin syndrome. With amphetamine toxicosis, secondary stimulation of certain body systems can result in significant clinical signs: GI (e.g., vomiting, diarrhea, hypersalivating), CNS (e.g., agitation, mydriasis, tremors, seizures), cardiovascular (e.g., tachycardia, hypertension), and respiratory (e.g., panting). Both clinical signs and treatment for amphetamine toxicosis are similar to SSRI toxicosis, and include IV fluids, cooling measures, sedation (e.g., with acepromazine or chlorpromazine), muscle relaxants, anticonvulsants, thermoregulation, blood pressure monitoring, and symptomatic/supportive care.

SLEEP AIDS

Sleep aids are often benzodiazepines or non-benzodiazepine hypnotics, and include drugs such as zolpidem (Ambien®) and eszopiclone (Lunesta®). These drugs work similarly to benzodiazepines (e.g., diazepam) as they potentiate GABA transmission, increasing frequency of chloride channel opening and resulting in inhibition of neuronal excitation. While these drugs result in sedation in humans, up to 40-50% of dogs ingesting toxic doses of sleep aids develop paradoxical CNS stimulation rather than expected depression. Clinical signs include CNS depression (e.g., depression, ataxia, weakness, paresis), CNS stimulation (e.g., hyperactivity, anxiety, agitation, panting, tremors), or other signs like nausea, vomiting, diarrhea, and hyperthermia. Treatment includes decontamination, activated charcoal, and for those patients demonstrating signs of CNS stimulation, the use of sedatives or anxiolytics. In patients exhibiting CNS stimulation, benzodiazepines (e.g., diazepam IV) should *not* be used, as they may worsen the symptoms. Rather, the use of phenothiazines (e.g., acepromazine, chlorpromazine) or barbiturates (e.g., phenobarbital IV) should be used instead. In severe cases of respiratory or cardiac depression, the use of the reversal agent, flumazenil, can be considered.

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS (NSAIDS)

NSAIDs are competitive inhibitors of prostaglandin synthesis (cyclooxygenase or "COX" inhibitors) and result in decreased prostaglandin, which is important for normal homeostatic function (including maintaining renal blood flow, maintaining mucous production in the stomach, etc.). Common OTC human NSAIDs include active ingredients such as ibuprofen and naproxen sodium. Examples of human NSAIDs include Advil®, Aleve®, certain types of Motrin®, etc. Common prescription veterinary NSAIDs can also result in toxicosis, particularly when available in the chewable, palatable formulation. Examples of veterinary NSAIDs include carprofen, deracoxib, etogesic, previcoxib, etc. With NSAID toxicosis, the GI tract, kidneys, CNS, and platelets can be affected. Cats and certain breeds of dogs (e.g., German shepherds) seem to be more sensitive to NSAIDs, and should be treated aggressively. With cats, severe acute kidney injury (AKI) is often more clinically seen with NSAID toxicosis at lower doses (as compared

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to dogs). With dogs, signs secondary to GI ulceration (e.g., vomiting, diarrhea, melena, hematemesis, etc.) are more commonly seen initially, followed by secondary AKI.

With NSAID toxicosis, it is important to keep in mind that each NSAID has a different toxic dose, margin of safety, half-life, and route of excretion, and an animal poison control should be contacted to identify what specific NSAID and toxic dose was ingested. For example, in dogs, ibuprofen results in GI signs at doses as low as 16-50 mg/kg, while severe GI signs may be seen at 50-100 mg/kg.[6] Renal compromise may be seen at doses of 100-250 mg/kg (resulting in potential AKI), and fatalities have been reported at doses > 300 mg/kg.[6] This differs tremendously from naproxen sodium (dogs), where severe clinical signs can be seen at doses as low as 5 mg/kg.[6] Clinical signs of NSAID toxicosis include anorexia, vomiting, hematemesis, diarrhea, melena, abdominal pain, lethargy, malaise, uremic halitosis, dehydration, etc. Treatment includes decontamination, the use of activated charcoal (often multiple doses due to enterohepatic recirculation, if appropriate), GI protectants (e.g., H_2 blockers, sucralfate), aggressive IV fluid therapy (to help maintain renal blood flow), anti-emetic therapy, and symptomatic and supportive care. With high doses, anti-convulsants may also be necessary if CNS signs develop.

PYRETHRINS AND PYRETHROIDS

Pyrethrins and their synthetic derivative, pyrethroids, are commonly found in household insect sprays and insecticides (e.g., permethrin, cypermethrin, cyphenothrin, etc.). Due to a cat's altered liver glururonidation metabolism, cats are significantly more sensitive to pyrethrins than dogs. While a precise toxic dose for cats is not well established, products containing greater than a 5-10% concentration of pyrethrins may lead to systemic toxicosis. The diluted amount commonly found in household insect sprays and topical flea sprays and shampoos is typically < 1%, and unlikely to result in toxicosis (even in cats). The more common route of exposure in cats is inappropriate application of a *concentrated* canine spot-on pyrethin/pyrethroid based insecticides (typically ~40-50% concentration) to cats. Cats that groom dogs following recent spot-on applications are also be at high risk for toxicosis; ideally, pets should be separated until the spot-on product has completely dried on the dog to prevent cat exposure. Signs of systemic toxicosis in cats include GI signs (e.g., hypersalivation, vomiting, nausea), neurologic signs (e.g., disorientation, anxiety, weakness, hyperexcitability, tremors, seizures) and respiratory (e.g., tachypnea, dyspnea) signs. Tremors are extremely responsive to parenteral methocarbamol (22-220 mg/kg, IV PRN to effect), a centrally acting muscle relaxant; oral absorption of methocarbamol is often much slower in onset of action. In general, tremors are less responsive to benzodiazepines (e.g., diazepam). Seizures may be controlled with phenobarbital (e.g., 4-20 mg/kg, IV PRN to effect), diazepam (e.g., 0.25-0.5 mg/kg, IV PRN to effect), other anticonvulsants, or general gas anesthesia. Dermal decontamination is crucial but should be performed after stabilization (e.g., obtaining IV access, administering methocarbamol IV, etc.). Dermal decontamination should be performed with a liquid dish detergent (e.g., Dawn, Palmolive). Additional treatment includes supportive care, thermoregulation, and blood glucose monitoring. Signs may persist for 1-3 days, depending on the animal. The prognosis is excellent with aggressive dermal decontamination and treatment.

Metropolitan New Jersey Veterinary Medicine Association
New Jersey

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INSECT BAIT STATIONS

Household ant and roach bait stations are rarely toxic, as the active ingredient is typically a low-concentration of three ingredients: abamectin (a macrocylic lactone derivative in the same family as ivermectin), hydramethylnon (an insecticide that rarely affects mammalian tissue) or fipronil. Certain breeds with the MDR-1 gene mutation (now known as the ABCB1-1Δ polymorphism), including collies, Border collies, old English sheepdogs, and collie-mixed breed dogs, may be more at risk when large amounts of bait stations are ingested. Typically, the plastic on the bait station is more of a problem, as it can result in GI signs or potentially foreign body obstruction (FBO), when ingested in large amounts.

HOUSEHOLD CLEANERS

Most surface cleaners are generally benign, and when ingested directly from the bottle, can result in minor GI signs. However, certain concentrated cleaners can be highly toxic or corrosive. Household bleach is a GI irritant, but "ultra" bleach can be corrosive, resulting in severe esophageal or upper GI damage. Concentrated lye products, toilet bowl cleaners, and oven cleaners are also corrosive, and immediate flushing out the mouth for 10-15 minutes should be performed prior to veterinary visit to minimize tissue injury. Appropriate pet-proofing (such as keeping toilet seats down or securing cleaners in a locked or elevated bathroom cabinet) are the easiest way to prevent this specific toxicosis.

BATTERIES

Battery ingestions occur quite frequently by dogs. This is often witnessed by the owner, or a chewed battery may be discovered by the owner. Often times, the pet owner may notice that the remote control is chewed on and the batteries are missing. When the casing for a battery is punctured, there is risk for alkaline or acidic material to leak out, resulting in severe ulceration to exposed tissues. The most common battery ingestion is of an alkaline dry cell battery (e.g., 9-volt, D, C, AA, AAA) or button/disc batteries. Alkaline dry cells (the majority of household batteries) contain potassium hydroxide or sodium hydroxide. When the compounds come in contact with tissue, liquefaction necrosis occurs, causing deeply penetrating ulcers. In addition, newer types of "disc shaped" batteries can allow an electric current to pass to the tissues of the GI tract as the battery is passed. This can result in a current-induced necrosis, resulting in tissue damage or even perforation of the oropharynx, esophagus, stomach or small intestine. Lithium button type batteries are the most dangerous, as one 3 volt battery can result in severe necrosis to the GI tract or esophagus within 15-30 minutes of contact. Finally, certain batteries contain heavy metals (e.g., mercury, zinc, cobalt, lead, nickel or cadmium). Heavy metal toxicity can occur, albeit rare, if the battery remains in the GI tract for more than 2-3 days.

With any type of battery ingestion, the pet owner should seek veterinary attention immediately. A thorough oral exam and physical exam should be performed. Oral ulcerations may not be present on physical examination for several hours, and the absence of oral ulcerations does not rule out severe underlying corrosive injury lower in the GI tract. The presence of black powdered material may be seen

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in the mouth, and occurs when dry cell batteries are punctured. The mouth should be thoroughly flushed and lavaged for 15-20 minutes with tepid tap water. A lateral abdominal radiograph (including the caudal esophagus in the chest) should be performed to evaluate the presence of the battery in the abdomen. Ideally, prompt removal should occur to prevent further corrosive injury. The use of endoscopy or surgery may be necessary. Emesis induction is not typically recommended, as corrosive injury may occur to the esophagus and oropharynx. Treatment includes removal of the battery, anti-ulcer medication (including H_2 blockers and sucralfate) for 5-7 days, a bland or high-fiber diet, and analgesic therapy if necessary.

SILICA GEL PACKS & FOOD OXIDIZER PACKS

Silica gel packs, while commonly ingested by pets, rarely result in toxicosis as they have a wide margin of safety (despite their labeling of "Do not eat"). When ingested in large amounts, they can potentially result in FBO; however, this is generally rare. These need to be differentiated from food oxidizer packs which contain small amounts of iron; food oxidizer packs are commonly found in human and pet food products (e.g., box of cookies, cereal, pasta containers, jerky treats, rawhide packs, etc.). The contents of a food oxidizer pack are typically brown or black in color (e.g., pellets, powder) and stick to a magnet; this is consistent with iron. As iron does not bind well to activated charcoal (AC), the use of AC is not indicated. Rather, with iron toxicosis, the use of antacids like milk of magnesia should be considered, which readily bind to heavy metals. In general, treatment is typically not necessary as these packs are very small; however, in a very small patient, outpatient treatment may be warranted as needed.

HYDROCARBONS

Hydrocarbons consist of chemicals containing a hydrogen and carbon group as their main constituents. Examples include liquid fuels such as kerosene, engine oil, tiki-torch fuels, gasoline, diesel fuels, paint solvents, wood stains, wood strippers, liquid lighter fluids, asphalt/roofing tar, etc. These are often referred to as "petroleum distillates" based on their viscosity, carbon chain length, and lipid solubility. It is *contraindicated* to induce emesis with hydrocarbon toxicosis due to the risks of aspiration pneumonia; due to the low viscosity of hydrocarbons, these compounds are more easily aspirated, resulting in respiratory injury and secondary infection. In general, hydrocarbons are GI tract irritants, but can also be irritants to the respiratory system (if inhaled), eyes, and skin also. Clinical signs include vomiting, nausea, tachypnea, and dermal or ophthalmic irritation. Typically, GI tract irritation is self-limiting. Patients should be treated with anti-emetic therapy, possible SQ fluid therapy (to assist in hydration), fasting (no food per os), and initiation onto a bland diet. Patients demonstrating any coughing, retching, or tachypnea post-ingestion should have chest radiographs performed to rule out aspiration pneumonia, of which treatment is supportive (e.g., oxygen therapy, IV fluids, antibiotic therapy, nebulization and coupage, etc.).

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FERTILIZERS

Fertilizers generally have a wide margin of safety, and result in mild GI signs when ingested directly. These typically contain natural elements (e.g., nitrogen, potassium, potash); sometimes, it may also contain an insecticide (typically a low concentration pyrethrin). Ingestion of grass that had a fertilizer applied to it previously rarely results in serious toxicosis; more serious clinical signs can be seen when the product is directly ingested (e.g., directly out of the bag). When appropriately applied or diluted, these chemicals typically wash into the soil after rainfall, resulting in low-risk to patients. What is key is to make sure that the compound was not mixed or does not contain more dangerous (but rarer) insecticides such as carbamates or organophosphates.

BONE OR BLOOD MEAL

Bone meal and blood meal are by-products from the meatpacking or animal feed industry; these are commonly used as "natural" and "organic" soil amendment products. Pet owners often use these more frequently (as compared to fertilizers) due to the "organic" nature; this has resulted in an increased exposure to animals. These are often considered low-level toxicities, but can result in FBO, severe pancreatitis, or GI tract irritation with ingestion. Pet owners should be questioned on whether these more benign meals have been mixed with more toxic agents (such as organophosphates [OPs] found in rose fertilizers), which result in severe toxicosis. Bone meal and blood meal are highly palatable to dogs and can result in unintentional, large ingestions. Tulip, daffodil and hyacinth bulbs are often "dusted" in bone meal when planted to fertilize and aid in repelling squirrels. The scent of bone meal may entice dogs to dig up newly planted bulbs and subsequently ingest both the potentially toxic bulb and bone meal. Large ingestions of bone meal can congeal into a solid ball or bezoar in the stomach, resulting in a FBO. Large ingestions of blood meal can congeal into a gelatinous FBO. Decontamination is recommended with recent large ingestions or with dogs with a prior history of pancreatitis. Radiographs should be performed to determine if the material has passed out of the stomach prior to emesis induction, and to evaluate for the presence of gastric contents or FBO. With massive ingestions demonstrating evidence of FBO, surgical intervention may be necessary (albeit rarely) if gastric lavage fails to decontaminate the patient. In general, decontamination and symptomatic and supportive care are all that is indicated.

DECONGESTANTS

Cold and flu medications (e.g., "Claritin-D") often carry decongestants such as pseudoephedrine (PSE) and phenylephrine (PE). The exact mechanism of how these drugs work is unknown but thought to stimulate alpha and beta-adrenergic receptors by releasing norepinephrine. Phenylephrine is typically considered to be less toxic than PSE as it is less bioavailable with oral ingestion. Clinical signs seen with decongestant ingestion include cardiac (e.g., tachycardia, hypertension, reflex bradycardia), CNS (e.g., mydriasis, agitation, trembling, seizures), and various miscellaneous signs (e.g., hyperthermia). With PSE, moderate to severe clinical signs can be seen at 5-6 mg/kg, while death has been reported at 10-12 mg/kg. With phenylephrine, similar clinical signs can be seen, although GI signs such as vomiting are the

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most common sign observed. Treatment includes decontamination (if appropriate), administration of one dose of charcoal with a cathartic, IV fluid therapy (to enhance urinary elimination), blood pressure monitoring, anti-emetics, sedatives/anxiolytics (e.g., acepromazine), muscle relaxants for tremoring (e.g., methocarbamol 22-100 mg/kg, IV PRN), anticonvulsants (e.g., phenobarbital 4-6 mg/kg, IV, PRN), and rarely, anti-hypertensives (e.g., hydralazine).

CONCLUSION

Pet owners should be appropriately educated on how to pet-proof the house, and be trained on what common household products and kitchen items are poisonous. Pet owners should also be appropriately educated on crate training to help minimize toxin exposure. Once a pet is exposed to a toxicant, it is imperative to determine if emesis is appropriate, and to understand when it may be contraindicated (e.g., symptomatic patient, delayed time since exposure, hydrocarbons, etc.). Knowledge of the underlying mechanism of action, the pharmacokinetics (including absorption, distribution, metabolism, and excretion), and the toxic dose of the toxicant are imperative in determining appropriate decontamination and therapy for the patient. **When in doubt, the** ASPCA Animal Poison Control Center should be consulted for toxic ingestions that veterinarians are unaware of.

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