

Common Pet Toxicants, Diagnosis and Treatment

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Many common pet toxicants are readily available around the house, garden or garage. Knowing these and understanding the underlying mechanism of action, pharmacokinetics, clinical signs seen and treatments is necessary to appropriate management of poisoned patients.

Pet Poison Helpline, an animal poison control that is available around the clock, recently reported its most common calls for canine toxicosis. More than 50% of the calls to Pet Poison Helpline were related to human medications. Top calls included:

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| Methylxanthines |
| Insect bait stations |
| Rodenticides |
| Fertilizers |
| Xylitol-containing products |
| Ibuprofen |
| Acetaminophen |
| Silica gel packs |
| Amphetamines (such as ADD/ADHD drugs) |
| Household cleaners |

In July, VPI® Pet Insurance released its top canine poisoning claims submitted over a five-year period and number of claims:

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| Drug reaction/accidental ingestion of drugs: 5,131 (including accidental overdoses of both human and pet medications as well as inadvertent reactions to prescribed medications) |
| Rodenticides: 4,028 |
| Methylxanthines: 3,661 |
| Plant toxicosis: 2,808 |
| Household cleaners: 1,669 |
| Metaldehyde: 396 |
| Insecticides/organophosphates: 323 |
| Heavy metal poisoning: 288 |
| Toad poisoning: 270 |
| Antifreeze: 213 |
| Walnuts: 100 |
| Alcohol: 75 |
| Strychnine: 28 |



As one can see, there is extensive overlap between these two lists. A brief review follows.

- **Human medicines.** NSAIDs such as human OTC products like ibuprofen and naproxen; acetaminophen; antidepressants; amphetamines which are used to treat attention deficit disorder (ADD) or attention deficit hyperactivity disorder (ADHD) and sleep aids topped the calls received at Pet Poison Helpline. NSAIDs can result in severe gastrointestinal (GI) ulceration and potent renal vasoconstriction, resulting in acute renal failure. Acetaminophen can result in hepatotoxicity and severe methemoglobinemia. Antidepressants, amphetamines, and sleep aids can result in a variety of signs, including sedation, agitation, tachycardia, hypertension, tremors, seizures and secondary hyperthermia. Aggressive supportive care and treatment is imperative with human medication toxicosis. As each drug contains different mechanisms of action and toxic ranges, Pet Poison Helpline should be consulted to identify appropriate treatment.
- **Rodenticides.** One of the most common mistakes made in veterinary toxicology is assuming that every green or blue rodenticide is a long-acting

anticoagulant (LAAC). Several different classes of rodenticides exist and are commonly mistreated with Vitamin K₁ therapy. Other types include bromethalin, zinc phosphide and cholecalciferol rodenticides. *Bromethalin* works by uncoupling oxidative phosphorylation in the brain and liver mitochondria, resulting in cerebral edema, with clinical signs seen as ataxia, decreased mentation, tremors, seizures, etc. *Phosphide* rodenticides result in the production of phosphine gas, which is also toxic to humans. When zinc phosphide combines with gastric acid or food, liberated phosphine gas is rapidly absorbed across gastric mucosa and distributed systemically. Clinical signs include severe GI (e.g., vomiting, bloat, abdominal pain, etc.), neurologic (e.g., tremoring, seizing) and pulmonary signs (e.g., pulmonary edema, tachypnea, etc.). More importantly, emesis—whether intentionally induced or occurring due to clinical signs—can result in poisoning the pet owner or the veterinary professional. Clinical signs of nausea and difficulty breathing have been reported in humans exposed to secondhand phosphine gas. Treatment with an antacid prior to emesis induction may help decrease the presence of phosphine gas. Also, emesis induction should always be performed in a well-ventilated area. *Cholecalciferols* are the most deadly and costly to pets, as they result in severe hypercalcemia with secondary mineralization of the kidneys and soft tissues. This results in acute and potentially chronic renal failure and must be treated aggressively with IV fluid therapy, calcium monitoring and administration of steroids, diuretics and bisphosphonates. Finally, first- and second-generation LAAC anticoagulants result in inhibition of Vitamin K epoxide reductase, resulting in inactivation of clotting factors II, VII, IX and X. Clinical signs and elevation in clotting factors, prothrombin (PT) or activated partial thromboplastin time (aPTT) are not seen for 48 hours. Treatment includes decontamination, measurement of PT/PTT 48 hours post-ingestion, or initiation of prophylactic treatment with Vitamin K₁ for a minimum of four weeks. A recheck PT should be performed 48 hours after the last administered dose; if prolonged, an additional two weeks of Vitamin K₁ therapy should be administered.

- **Methylxanthines.** These chemicals are found in chocolate, tea and coffee products, and can cause “stimulating” clinical signs of vomiting, diarrhea, agitation, tachycardia, tachypnea, tremors and seizures with large ingestions. As the concentration of methylxanthine varies between each product ingested (e.g., white chocolate contains minimal amounts while dark chocolate

contains large amounts), clinical signs can be seen at variable doses and should be calculated appropriately to determine if a toxic amount has been ingested or what therapeutics should be initiated.

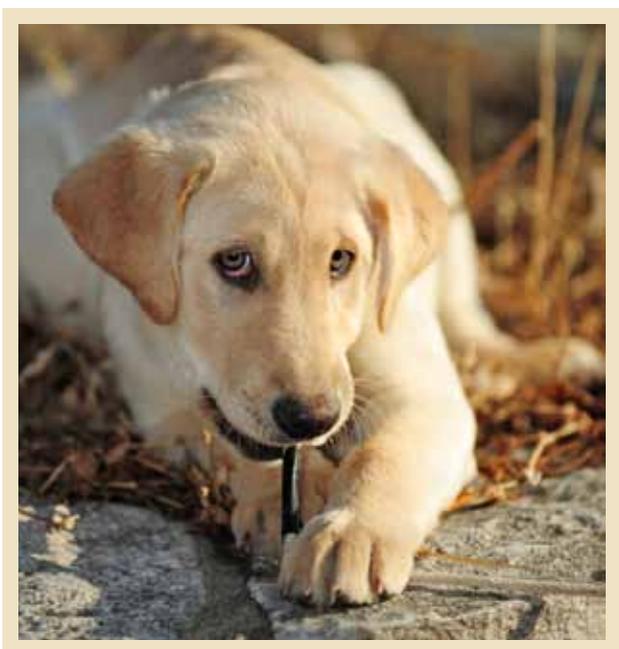
- **Plants.** As different plants have different mechanisms of action and levels of toxicosis, Pet Poison Helpline should be consulted when necessary for assistance in managing plant ingestions. While the majority of plant ingestions often just result in GI signs, some can be fatal. The most deadly plant is *Sago palm*, a warm weather plant that can cause acute hepatic failure. The prognosis is grave once clinical signs of liver failure have developed and long-term outcome is often poor. *Oleander*, which contains a cardiac glycoside, can result in profound cardiovascular signs (brady- or tachyarrhythmias), electrolyte abnormalities (e.g., hyperkalemia), GI signs (e.g., nausea, hypersalivation, vomiting) or central nervous system (CNS) signs (e.g., tremors, seizures). *Japanese yew*, which is commonly used as a landscaping shrub, results in profound GI, CNS and cardiovascular signs due to the toxic alkaloids called taxines. *Dieffenbachia* and *philodendron* contain insoluble calcium oxalate crystals which result in profuse pain to the oropharynx. This differs from soluble calcium oxalate-containing plants like Easter lilies, star fruit or rhubarb which can result in calcium oxalate deposition in the kidneys and secondary acute renal failure in cats or patients with underlying renal insufficiency. Certain spring bulbs such as daffodils, tulips, *Narcissus*, etc. can result in profuse GI signs and with large ingestions, cardiotoxicity or neurotoxicity.

- **Household cleaners.** Most surface cleaners are generally benign and when ingested directly from the bottle, can result in 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 of the mouth for ten minutes should be performed prior to the veterinary visit to minimize tissue injury. Appropriate pet-proofing such as keeping toilet seats down or securing cleaners in a bathroom cabinet are the easiest way to prevent this type of toxicosis.

- **Metaldehyde.** This slug and snail bait, commonly used in California and warm weather locations, results in “shake and bake” syndromes of agitation, tremors, seizures and secondary hyperthermia. The pellets are often radiopaque. Radiographs can be performed to evaluate for the presence of material within the GI tract. Aggressive supportive care, including decontamination (e.g., gastric lavage and enemas), treatment with anti-convulsants such as phenobarbital, muscle relaxants like methocarbamol and monitoring of hepatic function should be performed.
- **Insecticides/organophosphates.** Many insecticides are low-concentration pyrethrins or pyrethroids and generally only result in mild GI signs when directly ingested. However, some products such as rose or plant fertilizer/insecticide combinations may contain carbamates or organophosphates which competitively inhibit acetylcholinesterase and pseudocholinesterase. This results in acetylcholine accumulation at nerve junctions, resulting in severe clinical “SLUDGE” signs (e.g., salivation, lacrimation, urination, defecation, gastrointestinal). Gardeners often mix these insecticides/fertilizers with



additional bone or blood meal which is highly palatable to pets; the result is an increased amount of toxin ingestion. Aggressive therapy with 2-PAM and atropine is imperative, along with 24-hour supportive care.

- **Antifreeze.** Accidental or malicious poisoning with antifreeze is common as the public is generally well aware of the narrow margin of safety. As little as a tablespoon can result in severe acute renal failure in canines. While the antidote fomepizole (also known as 4-MP) is expensive, it is life-saving when administered within the first three to 12 hours of ingestion. Ethanol, which also competes with alcohol dehydrogenase thereby preventing metabolism of ethylene glycol into its more toxic metabolites, can also be used. Once a patient has already developed azotemia, the prognosis is generally poor to grave without hemodialysis.
- **Xylitol.** Sugarless gums, particularly those with xylitol listed within the first five active ingredients, can result in severe toxicosis within 15 minutes of ingestion. Ingestion of xylitol results in an insulin spike in non-primate species, resulting in severe hypoglycemia. As each piece of candy or gum may contain various amounts of xylitol ranging, on average, from 0.22 grams/piece to 1.0 grams/piece, it is imperative to calculate if a toxic dose has been ingested. Doses > 0.1 g/kg are considered toxic, and can result in hypoglycemia. Treatment includes STAT blood glucose monitoring and IV fluid therapy with dextrose supplementation as needed. Higher doses (> 0.5 g/kg) can result in acute hepatic necrosis and should be treated aggressively with hepatoprotectants (e.g., s-adenosylmethionine), IV fluids, anti-emetics and supportive care, including liver enzyme monitoring.

Veterinarians and pet owners can be prepared through education and prevention. The veterinarian can begin pet owner education at the very first wellness exam visit. Providing educational material in the form of medically accurate handouts that are easy to read and often have toxin identification pictures is an effective, efficient way of educating pet owners.

Secondly, provide appropriate counseling. Ask probing questions at every wellness visit which often indirectly educates pet owners about potential common toxins. In the emergency room, assessment of an animal should include the three “Ts”: trauma, toxins and ticks. Specifically inquire about any possible exposure to toxins such as Easter lilies (felines), grapes/raisins (canines), rat poison, etc. During history questioning, pet owners are often surprised that grapes and raisins are poisonous. Finally, educate pet owners on what to do in case

of a pet poisoning. Recommend that they pre-program their cell phone with your clinic's phone number, the emergency clinic's number and Pet Poison Helpline's phone number (1-800-213-6680) to provide immediate access to medical support during emergency situations.

Prevention is just as crucial. Take time to reiterate the importance of crate training, particularly with Labrador retrievers and mixed breed dogs as these breeds most commonly ingest poisons according to Pet Poison Helpline. Educate pet owners on how to adequately pet-proof their house and what to do at the time of ingestion of a possible poison. Here are some simple, effective tips:

- Hanging up a purse. Albuterol inhalers, prescription bottles, xylitol gum, zinc pennies and birth control are often found in a purse.
- Storing pet medications away from human medications. Pet Poison Helpline often receives phone calls from frantic pet owners who accidentally pillled their own medication to their pet.

- Keeping weekly medication containers out of reach. These plastic weekly pill holders look and sound like a dog rattle chew toy.
- Avoiding putting multiple days of medication in a plastic storage bag.
- Never leaving medication stuffed in a very palatable aid such as GREENIES® PILL POCKETS® unless you're ready to administer it immediately, as another pet often may ingest it instead.
- Keeping pills—particularly sleep aids, antidepressants, etc.—off the night table.



Contacting a veterinarian or Pet Poison Helpline for immediate advice is of the utmost importance, as the narrow window for decontamination is limited in veterinary medicine and is still the mainstay of therapy. The prognosis is better when a patient is rapidly decontaminated and treated aggressively, compared to waiting until clinical signs develop, where the prognosis is poorer and the cost—both emotionally, financially and physically to the owner and the pet—may be higher.

Lastly, bring to your client's attention the value and importance of pet insurance for emergencies such as poisonings. VPI® Pet Insurance policies cover emergencies and unexpected events, providing coverage for costly veterinary visits. No one plans on their pet being poisoned and in emergency situations prompt care and pet insurance coverage are of the utmost importance.



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