

TOP 20 TOXINS... AND HOW TO TREAT THEM!

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INTRODUCTION

Pet Poison Helpline, a 24/7 animal poison control located out of Minneapolis, MN, receives phone calls from both pet owners and veterinarians regarding toxicity cases from accidental or intentional misuse of over-the-counter (OTC) or prescription medications, common garden or outdoor toxins, and common household products. In this two hour session, the top 20 small animal toxins seen by Pet Poison Helpline will be reviewed.

In veterinary medicine, the primary treatment for toxicant exposure should be decontamination and detoxification of the 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. Decontamination can only be performed within a narrow window of time for most substances; therefore, it is important to obtain a thorough history and time since exposure. Decontamination categories may include ocular, dermal, inhalation, gastrointestinal (GI), forced diuresis, and surgical removal to prevent absorption or enhance elimination of the toxicant. For further review on decontamination and specific treatment, attendees are referred to a veterinary toxicology book for more detailed review.

SELECTIVE SEROTONIN RE-UPTAKE INHIBITORS (SSRIS)

Selective serotonin re-uptake inhibitors (SSRIs) are a class of medications that are commonly used in human medicine for depression. Common examples include drugs like fluoxetine (Prozac[®] in human beings; Reconcile[™] in veterinary medicine), citalopram (Celexa[®]), escitalopram (Lexapro[®]), paroxetine (Paxil[®]), and 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 sedation or central nervous system (CNS) stimulation, anorexia, and lethargy, even at therapeutic doses. Increases in levels of serotonin, even in small doses, may lead to serotonin syndrome. Clinical signs of serotonin syndrome include: CNS stimulation, vomiting, tremoring, seizures, hyperthermia (secondary to tremoring and seizuring), diarrhea, abdominal pain, and mydriasis. Treatment includes decontamination (ideally done at a veterinarian, due to the rapid onset of clinical signs), activated charcoal, hospitalization for sedation (e.g., with acepromazine or chlorpromazine), thermoregulation, IV fluid therapy, blood pressure and electrocardiogram (ECG) monitoring, and supportive and symptomatic care.

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 include dextroamphetamine and amphetamine (Adderall[®]), D-amphetamine (Dexedrine[®]), methamphetamine (Desoxyn[®]), and lisdexamfetamine (Vyvanse[®]). 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: CNS (e.g., agitation, mydriasis, tremors, seizures), cardiovascular (e.g., tachycardia, hypertension), GI (e.g., vomiting, diarrhea, hypersalivating), and respiratory (e.g., panting). The oral lethal dose is low for amphetamines, and ranges from 10-23 mg/kg. 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), 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., intravenous [IV] diazepam) 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 flumazenil, the reversal agent for benzodiazepines, can be considered.

GRAPES, RAISINS, AND CURRANTS

Grapes and raisins (*Vitis* spp) have been recently associated with development of acute renal failure (ARF) with ingestion. All types have been implemented with toxicosis, including organic grapes, commercial grapes, homegrown grapes, and seedless or seeded grapes. While the mechanism of toxicosis is unknown, there are several suspected hypotheses, including individual inability to metabolize certain components of the fruit (e.g., tannins, high monosaccharide content),¹ the presence of mycotoxins or pesticide residues on the fruit,¹ or salicylate-like chemicals within the grape or raisin. Common kitchen items also contain grapes, raisins, or currants in their active ingredient, including raisin bread, trail mix, chocolate-covered raisins, cereal with raisins, etc. Currently, grapeseed extract has not been associated with nephrotoxicity.¹ Treatment for grape and raisin ingestion includes aggressive decontamination as the first-line of therapy. Grapes and raisins seem to stay in the stomach for a prolonged period of time, and are not rapidly broken down or absorbed from the GI tract; hence, delayed emesis induction even several hours post-ingestion can still be initiated to maximize decontamination methods. One dose of activated charcoal can also be administered to prevent absorption of the unknown nephrotoxin. As there is no current veterinary peer-reviewed, scientific published toxic dose of grapes and raisins, all ingestions should be treated as potentially idiosyncratic and be appropriately decontaminated and treated. Initially, vomiting may be observed within the first 24 hours of ingestion.¹ Within the next 12-24 hours, clinical signs of lethargy, dehydration, vomiting, diarrhea, anorexia, abdominal pain, uremic breath, and diarrhea may be seen.¹ Azotemia may develop within 24 hours, with hypercalcemia and hyperphosphatemia occurring first.¹ Oliguria and anuria may develop 48-72 hours post-ingestion,¹ at which point the prognosis is poorer. Treatment includes decontamination, aggressive IV fluid therapy, anti-emetics, blood pressure and urine output monitoring, and serial blood work monitoring (q. 12-24 hours). In severe cases, hemodialysis or peritoneal dialysis may be necessary. Asymptomatic patients that have been adequately decontaminated and survive to discharge should have a renal panel and electrolytes monitored 48-72 hours post-ingestion. Overall, the prognosis is fair to poor, depending on time to decontamination, response to therapy, and prevalence of oliguria or anuria. Overall, 50% of dogs that ingest grapes and raisins never develop clinical signs or azotemia, making the prognosis fair to good.¹

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 ARF is often more clinically seen with NSAID toxicosis at lower doses (as compared to dogs). With dogs, signs secondary to GI ulceration (e.g., vomiting, diarrhea, melena, hematemesis, etc.) are more commonly seen initially, followed by secondary ARF.

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 helpline 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.² Renal compromise may be seen at doses of 100-250 mg/kg (resulting in potential ARF), and fatalities have been reported at doses > 300 mg/kg.² This differs tremendously from naproxen sodium (dogs), where severe clinical signs can be seen at doses as low as 5 mg/kg.² With naproxen, experimental canine doses of 22 mg/kg orally once a day for 3 days have resulted in perforation of the GI tract with secondary septic peritonitis occurring.

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., H₂ 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.

XYLITOL

Xylitol is a natural sweetener found in small quantities in certain fruit. Xylitol has gained recent popularity because it is sugar-free, and is often found in diabetic snacks, foods, baked foods, mouthwashes, toothpastes, chewing gum, mints, candies, and chewable multivitamins.³ Sugarless products, particularly those with xylitol listed within the first five active ingredients, can result in severe toxicosis within 15-30 minutes of ingestion. Ingestion of xylitol results in an insulin spike in non-primate species, resulting in severe hypoglycemia. Many pieces of candy and gum (e.g., Orbit™, Trident™, Ice Breakers™) contain various amounts of xylitol ranging, on average, from 0.22 grams/piece to 1.0 grams/piece. Unfortunately, not all sources are disclosed by the company (e.g., how many grams of xylitol may be in each piece of gum) due to a proprietary nature. With xylitol toxicosis, it is imperative to calculate whether a toxic dose has been ingested. Doses > 0.1 g/kg are considered toxic and result in profound, sudden hypoglycemia from insulin stimulation.³ Higher doses (> 0.5 g/kg) of xylitol have been associated with acute hepatic necrosis. Clinical signs of xylitol toxicosis include lethargy, weakness, vomiting, collapse, anorexia, etc. When hepatotoxic doses are ingested, clinical signs and clinicopathologic findings may include melena, icterus, increased liver enzymes, diarrhea, hypoglycemia, hypocholesterolemia, decreased BUN, hypoalbuminemia, etc. When presented a patient that has ingested a toxic amount of xylitol, a blood glucose should be checked immediately upon presentation; if hypoglycemic, a bolus of 1 ml/kg of 50% dextrose, diluted with an additional amount of 0.9% NaCl (in a 1:3 ratio) should be given IV over 1-2 minutes. Emesis induction should not be performed until the patient is euglycemic. Keep in mind that activated charcoal does *not* reliably bind to xylitol, and is not routinely recommended for xylitol toxicosis. Hypoglycemic patients should be hospitalized for IV fluid therapy [supplemented with dextrose (2.5 to 5% dextrose, CRI, IV)] for approximately 24 hours, and frequent blood glucose check should be performed every 1-4 hours. For patients ingesting a hepatotoxic amount of xylitol, the use of hepatoprotectants (e.g., SAME), anti-emetics, and supportive care (including frequent liver enzyme monitoring) are warranted.

INSECT BAIT STATIONS

Household ant and roach bait stations are rarely toxic, as the active ingredient is often a low-concentration of abamectin (a macrocyclic lactone derivative in the same family as ivermectin). 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.

SILICA GEL 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. Some types – typically oxygen absorbers found in beef jerky or rawhide bags - may contain iron; this can potentially result in iron toxicosis in small dogs or when ingested in large amounts. The powder within these oxygen absorbers is often black in color and magnetic. Treatment for iron toxicosis includes antacid therapy (e.g., milk of magnesia), symptomatic supportive care, monitoring blood iron levels, and potential chelation (in severe cases). The use of activated charcoal is not warranted with iron toxicosis, as it does not reliably bind to heavy metals.

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 secured 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 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 H₂ blockers and sucralfate) for 5-7 days, a bland or high-fiber diet, and analgesic therapy if necessary.

FIRE STARTER LOGS

Fire starter logs typically do not pose a “toxicosis” risk, but rather a FBO risk. Most types (e.g., Duraflame[®]) are made of compressed sawdust and wax, and do not break down in the stomach, resulting in a FBO. Rarer types of fire starter logs may contain heavy metals to provide a “color sparkle” to the fireplace. With recent ingestion, emesis induction should be performed to prevent FBO. If unknown ingestion or prolonged ingestion has occurred, abdominal radiographs should be performed to evaluate for the presence of gastric contents or FBO. If the material has passed out of the stomach, the use of a high-fiber diet, anti-emetic therapy, and careful monitoring (based on clinical signs, radiographic evidence of obstruction, etc.) should be performed. With massive ingestions demonstrating evidence of FBO, surgical intervention may be necessary, albeit rare.

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.).

FERTILIZERS

Fertilizers generally have a wide margin of safety, and result in mild GI signs when ingested directly. 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.

BONE OR BLOOD MEAL

Bone meal and blood meal are by-products from the meatpacking industry that are widely utilized as soil amendment products, fertilizer components, or as deer, rabbit and wildlife repellants. Bone or blood meal are “organic” compounds, and with the increased use of organic products in lawn and gardening, have resulted in increased exposure opportunities for animals. These are often considered low-level toxicities, but can result in FBO, severe pancreatitis, or GI tract irritation with ingestion. A thorough history must be obtained from the pet owner, as these products are often 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. In general, decontamination and symptomatic and supportive care are indicated.

RODENTICIDES (BROMETHALIN, ZINC PHOSPHIDE, CHOLECALCIFEROL, LONG-ACTING ANTICOAGULANTS)

One of the most common mistakes seen in the field of veterinary toxicology is assuming that each green or blue rat or mouse poison is a long-acting anticoagulant (LAAC) rodenticide. Several different classes of rodenticides also exist, and are commonly mistreated with Vitamin K₁ therapy inappropriately. Other types of rodenticides 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 toxic to humans also. When zinc phosphide combines with gastric acid (or the presence of food!), liberated phosphine gas is rapidly absorbed across gastric mucosa and distributed systemically, where it exerts its toxic effect. Clinical signs include severe GI signs (e.g., vomiting, bloat, abdominal pain, etc.), CNS signs (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 to the pet owner or the veterinary professional. Clinical signs of nausea and difficulty breathing have been reported in humans exposed to secondary 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 (e.g., opening the car window if the patient vomits or inducing emesis outside or in a well-ventilated area). *Cholecalciferols* are the most deadly – and costly – to pets, as it results in severe hypercalcemia with secondary mineralization of the kidneys and soft tissues. This results in ARF and potentially chronic renal failure, and must be treated aggressively with IV fluid therapy, calcium monitoring, and administration of steroids, diuretics, and bisphosphonates. Lastly, 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 prophylactic treatment with Vitamin K₁ for a minimum of 4 weeks. A recheck PT should be performed 48 hours after the last administered dose; if prolonged, an additional 2 weeks of Vitamin K₁ therapy should be administered.

PLANTS

As different plants have different mechanisms of action or levels of toxicosis, Pet Poison Helpline should be consulted for plant ingestions that veterinarians are unaware of. While the majority of plants often just result in GI signs, some plant ingestions can be fatal. The most deadly plant is sago palm, which is found in warm weather locations (e.g., Southern USA), and can result in acute hepatic failure. (See below). 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 CNS signs (e.g., tremors, seizures). Japanese yew, which is commonly used as a landscaping shrub, results in profound GI, CNS, and cardiovascular signs also, due to the toxic taxins (alkaloids). *Dieffenbachia* and *Philodendron* (commonly known as familiar houseplants: mother-in-law's tongue or dumb cane), contain insoluble calcium oxalate crystals which result in profuse pain to the oropharynx. This differs from soluble calcium oxalate-containing plants (e.g., star fruit, rhubarb, etc.) which can potentially result in calcium oxalate deposition in the kidneys and secondary ARF (particularly in patients with underlying renal insufficiency). Certain spring bulbs (e.g., daffodils, tulips, *Narcissus*, etc.) can result in profuse GI signs, and with large ingestions, cardiotoxicity or neurotoxicity.

Lilies

The common Lily plant (from the *Lilium spp.* and *Heimerocallis spp.*) is often found in gardens, floral arrangements, or as fresh cuttings. These beautiful, fragrant flowers are known as the common Easter lily, tiger lily, Japanese show lily, stargazer lily, rubrum lily, and day lily.⁶ All parts of the plant, including the pollen, are toxic to cats, and result in severe ARF. As little as 1-2 leaves or petals, even the pollen, can result in ARF, and clinical symptoms are typically seen within hours. Clinical signs include early onset vomiting, depression, and anorexia, which progresses to anuric ARF in 1-3 days.⁶ Clinicopathologic testing reveals severe azotemia, epithelial casts (12-18 hrs post-ingestion) on urinalysis, proteinuria, and glucosuria.⁶ Treatment includes aggressive decontamination and IV fluid therapy for approximately 48 hrs. The use of subcutaneous (SQ) fluid therapy is not sufficient. While rarely performed in veterinary medicine, the use of peritoneal or hemodialysis has been successful in anuric ARF cases. With treatment, the prognosis is good if treatment is initiated early and

aggressively. Adequate decontamination (with emesis induction and activated charcoal) is of the utmost importance. If aggressive IV fluid therapy is initiated *within* 18 hours, the overall response to therapy is good. However, if treatment is delayed beyond 18-24 hours, or anuria has already developed, the prognosis is grave.⁶

Sago palm

Sago palms are naturally found in tropical/subtropical environments; they are also used as ornamental Bonsai houseplants. These palms are members of the Order Cycadaceae; genera *Cycads*, *Macrozamia*, and *Zamias*.⁷ Examples of the cycad family include Cycad (*Cycas cirinalis*), Japanese cycad (*Cycad revolute*), Coontie plant (*Zamia pumila*), and Cardbord palm (*Zamia furfuracea*).⁷ All parts of sago palm are considered poisonous, with the seeds (nuts) being the most toxic part of the plant.⁷ Sago palm contains cycasin, which is the primary active toxic agent resulting in hepatotoxicity. Ingestion results in acute GI signs (e.g., vomiting, diarrhea, hypersalivation) within 15 minutes to several hours after ingestion. Neurologic signs (e.g., weakness, ataxia, seizures, tremors, etc.) and severe acute, hepatic necrosis can be seen within 2-3 days post-ingestion. Clinical signs include vomiting, diarrhea, generalized malaise, anorexia, ascites, abdominal pain, icterus, and melena. Aggressive decontamination and treatment should be initiated. Baseline blood work and coagulation parameters should be monitored. Antiemetics, anticonvulsants, Vitamin K₁, hepatoprotectants (e.g., SAME), and broad spectrum antibiotic therapy is warranted. The use of N-acetylcysteine can also be used as a glutathione source. The prognosis is grave once clinical signs of liver failure have developed, and long-term outcome is poor as the potential for chronic liver disease and underlying potential myocardial injury exists.

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.

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