POISONOUS PLANT DANGERS TO DOGS AND CATS **TOXICOLOGY**

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Plant ingestions comprise a significant volume of small animal calls to the ASPCA Animal Poison Control Center (APCC) in Urbana, Illinois, and clinical cases presenting to the emergency clinic and general practitioner. While the majority of plant toxicosis typically results in mild gastrointestinal (GI) signs, a few plants can be life-threatening. Three particularly dangerous plants include lilies (cats), blue-green algae (dogs), and sago palm (dogs). For this reason, veterinary professionals need to be aware of the range in toxicosis of certain plant toxins. It is also important that veterinary professionals be able to rapidly and accurately identify certain common plants. When in doubt, the plant should be confirmed by a florist, botanist, master gardener (available at ASPCA APCC), or expert.

Insoluble Calcium Oxalates (Dieffenbachia/Philodendron)

According to the ASPCA APCC, the most common plant exposure is to the *Araceae* plant family.[1] These plants contain insoluble calcium oxalate crystals, and include the *Dieffenbachia* family of plants. These are common houseplants, as they require little water or light, and can survive in office conditions. Other types of insoluble oxalate containing plants include the following:[1]

- Arrowhead vine
- Calla lily
- Devil's ivy
- Dumbcane
- Elephant's ear
- Mother-in-law's tongue
- Peace lily
- Philodendron
- Pothos
- Sweetheart vine
- Umbrella plant

The plants contain needle sharp crystals, which are often arranged in bundles called raphides.[1] When dogs or cats bite or chew into the plant, it releases the crystals, resulting in acute, profuse pain to the oropharynx. Clinical signs of insoluble calcium oxalate plant toxicosis include hypersalivation, pawing at the mouth or muzzle, anorexia, vomiting, and edema of the lips, tongue, and oropharynx may be seen.[1] Very rarely, dyspnea and upper airway swelling can be seen secondary to severe inflammation and swelling of the laryngeal area. If ocular exposure occurs (rare), severe photophobia, pain, and conjunctival swelling can occur. While clinical signs may appear to be dramatic to the pet owner, signs are primarily localized to the oropharynx and generally are self-limiting. Treatment can potentially be done at home by the pet owner, and includes removal of the plant, flushing of the mouth (if possible), and offering small amounts of palatable fluid (e.g., canned tuna water, milk, yogurt, chicken broth, etc.) to flush the crystals from the mouth.[1] For more severe clinical signs that present to the veterinarian, the use of anti-emetics, fluid therapy (e.g., subcutaneous [SQ] or intravenous [IV]), or analgesics may be necessary. Atropine is *not* recommended for the hypersalivation.

Soluble Calcium Oxalates

A similar-sounding plant is the soluble oxalate–containing plant. These plants contain oxalic acid and oxalate salts, and must be differentiated from the plants above. Some examples of soluble calcium oxalate–containing plants include star fruit, common or garden rhubarb, and the shamrock plant.[2] This plant toxicosis is less commonly seen in small animals, and is generally considered more of a concern in large animals (that are chronically grazing on these plants).[2] That said, if this type of plant is ingested in large enough quantities by small animals, it can result in toxicosis.

Soluble calcium oxalates are present in varying degrees in all parts of the plant. For example, rhubarb stems are edible, but the leaves are not.[2] When soluble oxalate salts are absorbed from the GI tract, they bind with systemic calcium, resulting in an acute hypocalcemia.[2] The accumulation of calcium oxalate crystals then potentially can result in nephrosis and acute kidney injury (AKI).[2] While the likelihood of AKI is rare from soluble oxalate–containing plants, there is no known toxic dose reported in small animals. Dehydrated patients or those with underlying renal insufficiency may be more at risk for toxicosis, and should be treated more aggressively.[2] Clinical

signs include hypersalivation, anorexia, vomiting, diarrhea, lethargy, weakness, and tetany/tremors (secondary to hypocalcemia).[2] Once AKI has developed, signs of pu/pd, oliguria, oxaluria, hematuria, etc., may be seen 24–36 hours post-ingestion. Treatment for large ingestions includes decontamination (e.g., emesis induction, one dose of activated charcoal), fluid therapy, clinicopathologic monitoring (e.g., for hypocalcemia, oxaluria, azotemia, etc.), anti-emetic therapy, and symptomatic supportive care.

Lilies

The common "true" lily (from the *Lilium spp.* and *Hemerocallis spp*.) is often found in gardens, floral arrangements, or as fresh cuttings. These beautiful, fragrant flowers are known as the common Easter, tiger, Japanese show, stargazer, rubrum, and day lily.[3] All parts of the plant, including the pollen and water in the vase, are toxic to cats, and result in severe AKI. As little as 2–3 leaves or petals (even the pollen or water from the vase) can result in AKI, and clinical symptoms are typically seen within hours. Clinical signs include early onset vomiting, depression, and anorexia, which progresses to anuric AKI in 1–3 days.[3] Clinicopathologic testing reveals severe azotemia, epithelial casts (12–18 hrs post-ingestion) on urinalysis, proteinuria, and glucosuria.[3] Treatment includes aggressive decontamination (e.g., emesis induction, administration of one dose of activated charcoal), GI support (e.g., anti-emetics, H_2 blockers, etc.), and IV fluid therapy for approximately 48–72 hours (or until resolution of azotemia). The use of SQ fluid therapy is generally not sufficient for the treatment of lily toxicosis. While rarely performed in veterinary medicine, the use of peritoneal or hemodialysis has been successful in anuric AKI cases. With treatment, the prognosis is good if treatment is initiated early and aggressively. Adequate decontamination 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.[3]

Blue-Green Algae

Cyanobacteria (also known as blue-green algae) are one of the few toxicants that can result in *sudden* death in several species (e.g., humans, dogs, livestock, etc.). This microscopic bacteria is often found in nutrient-rich freshwater or brackish bodies of water, and grows more readily during hot, humid, stagnant weather conditions. While the *majority* of types of algae are non-toxic, it is very physically difficult to identify which type is toxin without diagnostic analysis. Blue-green algae typically appear as floating mats or blooms on the surface of the water. Blue-green algae can produce two toxins:[4]

- Microcystins (hepatotoxic, can result in acute liver failure along with clinical signs of lethargy, anorexia, vomiting, melena, diarrhea, pallor, hepatic encephalopathy, jaundice, seizures, and shock).
- Anatoxins (neurotoxicant, can result in SLUDGE-like signs, including salivation, lacrimation, urination, defecation, gastrointestinal signs, tremors, paralysis, seizures, cyanosis, etc.).

As this toxicant has a very narrow margin of safety, even minute ingestions can result in severe toxicosis or fatal poisonings. Depending on what type of clinical signs are seen (e.g., from either the microcystins or the anatoxins), treatment may include gastric lavage, IV fluid therapy (e.g., crystalloids, colloids), anti-emetics, blood glucose and clinicopathologic monitoring, glucose supplementation, anti-convulsants, muscle relaxants, antibiotics, atropine (if SLUDGE signs are present), vitamin K_1 or plasma transfusion administration (if coagulopathic), hepatoprotectants (e.g., SAM-e), oxygen therapy, and symptomatic supportive care. Unfortunately, the prognosis for this particular plant toxicant is poor and requires aggressive, 24/7 care.

Spring Flowers

Certain spring bulbs (e.g., daffodils, tulips, *Narcissus*, etc.) can result in profuse GI signs, and with large ingestions, cardiotoxicity or neurotoxicity. The most toxic part of these spring plants is the bulb. Tulips or hyacinths contain allergenic lactones tuliposides A and B, with tuliposides being most concentrated in the bulbs. Tulip and hyacinth bulbs also contain calcium oxalate crystals (see above). With tulip and hyacinth toxicosis, clinical signs of vomiting, hypersalivation, depression, and diarrhea may be seen. With large ingestions, tachycardia, dyspnea, and skin irritation may be seen (rare). Another common spring bulb is the daffodil (*Narcissus*). With ingestion, GI signs (e.g., hypersalivation, vomiting, diarrhea) may be seen; with large ingestions, hypotension, CNS signs (tremors, seizures), and cardiotoxicity (arrhythmias, tachycardia, etc.) are reported (but rarely seen). In general, treatment for spring bulb toxicosis is symptomatic and supportive and includes decontamination, fluid therapy (e.g., SQ or IV), anti-emetics, and possible abdominal radiographs to rule out foreign body obstruction or other underlying disease. In massive or severe cases, the use of blood pressure and electrocardiogram (ECG) monitoring, anticonvulsant therapy, and potentially anti-arrhythmic therapy is warranted.

Cardiac Glycosides (Foxglove, Oleander, Lily of the Valley, Kalanchoe)
Cardiac glycoside–containing plants pose a potential life-threatening toxicosis to animals; that said, toxicosis has been reported to be more severe in large animals (who are chronically grazing on plants) as compared to dogs or cats. Based on this author's clinical experience, this type of plant is less commonly seen in small animal medicine, but still poses a potentially significant risk with ingestion. Cardiac glycoside plants contain naturally occurring cardiotoxic cardenolides or bufadienolides, which interfere with the Na-K pump mediated by ATPase. This results in increased intracellular sodium and decreased intracellular potassium.[5] Examples of cardiac glycoside plants include[5]

- Dogbane
- Foxglove
- Giant milkweed
- Kalanchoe
- Lily of the valley
- Milkweed
- Oleander
- Star of Bethlehem

The toxins within these plants are similar to digitalis, and the degree of toxicity varies with the particular plant, part of the plant, and amount consumed.[5] All parts of the plant are generally considered toxic—even the water in the vase has been reported to cause toxicosis.[5] GI signs (e.g., nausea, hypersalivation, vomiting), profound cardiovascular signs (e.g., brady- or tachyarrhythmias, AV block, asystole), electrolyte abnormalities (e.g., hyperkalemia), or central nervous system (CNS) signs (e.g., mydriasis, tremors, seizures) may be seen.[5] Treatment includes decontamination, if appropriate, along with ECG and blood pressure monitoring. Clinicopathologic testing should be performed to evaluate for the severity of hyperkalemia and azotemia (which can be seen due to severe bradycardia and decreased cardiac output, albeit rare). The use of IV fluids, anti-emetics, and anti-arrhythmics are warranted. The antidote, digoxin-specific Fab fragments can be considered in severe, life-threatening cases (rare); however, due to the cost, its use is often precluded.

Mushrooms
While there are thousands of species of mushrooms in North America, less than 100 are poisonous; these are most commonly of the *Amanita* species. Unfortunately, mushrooms are very difficulty to identify, and unless you are a mushroom expert/hunter or mycologist, then you should *never* eat a wild mushroom. Mushrooms sold in large-chain grocery stores are safe and considered non-toxic to dogs.

There are 5 main types of mushrooms that are poisonous, and they all work by different ways:

- The most dangerous type of mushroom contains amanitin toxins, which results in severe gastrointestinal signs (within 6–24 hours), a "false recovery" period (where your dog appears to get better), and then severe liver failure (at 36–48 hours post-mushroom exposure). Kidney failure can also develop in the end stages. Examples of these types of deadly mushrooms include *Amanita, Galerina, Lepiota, A. phalloids* (death cap, death angel), and *A. ocreata.*
- Another type of mushroom contains muscarine and causes profuse SLUDE signs (e.g., salivation, lacrimation, urination, diarrhea) and neurologic signs. They work somewhat similarly to the organophosphate and carbamates chemicals. Examples of these types of mushrooms include *Inocybe spp.* and *Clitocybe dealbata.*
- One type of mushroom contains muscimol and ibotenic acid, and causes profuse signs like ataxia, sedation, and even tremors or seizures. Examples of these types of mushrooms include *Amanita muscaria* and *A.pantherina.*
- The false morel (*Gyromitra spp.*) causes profuse vomiting and diarrhea and is generally not fatal. Rarely, it can cause seizures.
- Some types of mushrooms just cause gastrointestinal irritation (e.g., vomiting, diarrhea) and are rarely life threatening when ingested. Signs can be seen in 1–6 hours, and generally resolve after 1–2 days. These types of mushrooms include the following types: *Agaricus, Boletus, Entoloma.*
- Hallucinogenic mushrooms aren't life-threatening and rarely need treatment. That said, signs of ataxia, acting abnormal, howling, nystagmus, and hyperthermia can be seen when dogs ingest them. These types of mushrooms include the following types: *Psilocybe, Conocybe, Gymnopilus spp.*

Volunteers at the National American Mycological Association (http://www.namyco.org/toxicology/index.html) may also help identify the mushroom for you (via image). If this is not readily available, all mushroom ingestions should be treated. As mushrooms are difficult to identify, treatment is based on "worst case scenario" (just in case it is *Amanita spp*.). As a result, treatment includes inducing vomiting (if appropriate), charcoal administration (to bind the poison from the stomach and intestines), anti-vomiting medication, and depending on what type of clinical signs are seen, anti-seizure medication, muscle relaxants, atropine, and symptomatic supportive care.

Sago/Cycad Palm

Sago palm, which are naturally found in tropical/subtropical environments (e.g., SE, S, SW United States) are life threatening to dogs and cats when ingested. Unfortunately, toxicosis can now be seen throughout North America; that's because these plants have grown in popularity and are now commonly sold as ornamental Bonsai houseplants. These plants are members of the Order Cycadacae; genera *Cycads, Macrozamia,* and *Zamias*.[6] Examples of the cycad family include[6]

- Cycad (*Cycas cirinalis*)
- Japanese cycad (*Cycad revolute*)
- Coontie plant (*Zamia pumila*)
- Cardboard palm (*Zamia furfuracea)*

All parts of sago palm are considered poisonous, with the female plant (e.g., seeds) being the most toxic part of the plant.[6] This plant 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 (AHN) 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 (if appropriate) and treatment should be initiated. Ideally, baseline blood work and coagulation parameters should be monitored. Treatment includes fluid therapy (e.g., IV crystalloids, colloids), anti-emetics, hepatoprotectants (e.g., SAMe), vitamin K_1 or plasma transfusion (if coagulopathic), anticonvulsants, and broad-spectrum antibiotic therapy (if in fulminant liver failure). The use of N-acetylcysteine (NAC) can also be used as a glutathione source. Unfortunately, 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 other damage exists.

Conclusion

While the majority of plant ingestions in small animals often just result in GI signs, some plant ingestions can result in significant clinical signs and can even be fatal without treatment. As different plants have different mechanisms of action or levels of toxicosis, ASPCA Animal Poison Control Center (888-426-4435) should be consulted for plant ingestions that veterinarians are unaware of.

References

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