

TOXIC PLANTS

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Insoluble calcium oxalate plants are very commonly kept as houseplants. Some of the more common ones include: *Aglaonema modestum* - Chinese evergreen, *Alocasia antiquorum* - Elephant's ear, *Anthurium* spp. - Flamingo plant, *Arisaema triphyllum* - Jack-in-the-pulpit, *Caladium* spp., *Dieffenbachia* spp. - Dumb cane, *Monstera* spp. - Split-leaf or lacy-leafed philodendron or Swiss cheese plant, *Philodendron* spp. and *Spathiphyllum* spp. - Peace Lily. The calcium oxalate crystals look like needles, and when the plant is damaged these crystals embed themselves in the oral cavity. Clinical signs include hypersalivation, vomiting and possible swelling of the pharynx (obstruction is rare). Treatment includes milk or water to rinse mouth. Clinical signs usually resolve within 24 hours with no lasting effects.

Dracaena (*Dracaena* spp) is a common house plant genus that includes the corn plant, dragon tree, money tree and lucky bamboo. It contains various steroidal saponins and glycosides. Clinical signs include vomiting (possibly with blood), hypersalivation, anorexia, depression, and ataxia. In addition, cats may show dilated pupils, dyspnea, and tachycardia. Treatment is symptomatic and supportive.

Poinsettias (*Euphorbia pulcherrima*) are overrated as a toxic plant. They do contain diterpenoid euphorbol esters in their sap which can cause vomiting.

Hydrangeas (*Hydrangea* spp.) contain hydrangin, a cyanogenic glycoside. However, in dogs and cats, GI signs predominate.

Bulb Plants include tulips (*Tulipa* spp.), daffodils, narcissus, amaryllis (*Narcissus* spp.) and hyacinths (*Hyacinthus* spp.). All parts of the plant are toxic, but the bulb is the most toxic. Ingestion of the flower or stem can cause vomiting. Ingestion of the bulbs can cause hemorrhagic gastroenteritis and neurologic signs.

Sago palms (*Cycas* and *Macrozamia* sp.) are ornamental plants found in tropical to subtropical climates, but they can also be grown as houseplants. There are three toxins in cycads. Cycasin is thought to be responsible for the hepatic and gastrointestinal signs. The seeds contain the highest amount of cycasin, but the entire plant is toxic. Cycasin causes centrilobular and midzonal coagulative hepatic necrosis along with GI hemorrhage. GI signs begin within a day and laboratory values (ALT, bilirubin, Alk Phos) become abnormal in 24 to 48 hrs. Mortality rate is about 30%. Treatment is emesis, followed by repeated doses of activated charcoal. Monitor liver enzymes for 48 hours, or until levels return to normal. Seizures and tremors may be controlled with diazepam. Blood or plasma transfusions may be

necessary if coagulopathies develop. Prognosis is guarded in cases where the animal is already showing signs.

Lilies of the *Lilium* and *Heimerocallis* sp. have been shown to cause acute renal failure in the cat. Some examples include: Easter lilies (*L. longiflorum*), tiger lilies (*L. tigrinum*), rubrum or Japanese showy lilies (*L. speciosum*, *L. lancifolium*), and day lilies (*H. species*). The toxic principle is unknown, but is known to be water soluble. Even minor exposures (a few bites on a leaf, ingestion of pollen, etc.) may result in toxicosis, so all feline exposures to true lilies should be considered potentially life-threatening and should merit aggressive clinical intervention. Affected cats often vomit within a few hours of exposure, but the vomiting usually subsides after a few hours, during which time the cats may appear normal or may be mildly depressed and anorexic. Within 24 to 72 hours of ingestion, oliguric to anuric renal failure develops, accompanied by vomiting, depression, and anorexia. Elevations in blood urea nitrogen (BUN), creatinine, phosphorus and potassium are detectable as early as 12 hours post ingestion. Casts, proteinuria, glucosuria, and isosthenuria are usually detectable on urinalysis within 24 hours of ingestion, reflecting lily-induced damage to renal tubular cells. In severe cases, death or euthanasia due to acute renal failure generally occurs within 3 to 6 days of ingestion. Treatment of lily cats includes decontamination (emesis, one dose activated charcoal with cathartic) and fluid diuresis at twice maintenance for 48 hours. If treatment is started within the first 18 hours after exposure, prognosis is good. Delaying treatment beyond 18 hours frequently results in death or euthanasia due to severe renal failure.

Cardiac glycoside containing plants include oleander (*Nerium oleander*), foxglove (*Digitalis purpurea*), and lily-of-the-valley (*Convallaria majalis*). The glycosides are rapidly absorbed from the GI tract and they inhibit the Na^+/K^+ ATPase pump, with resultant increase in intracellular sodium. The elevated intracellular Ca^{++} increases contractility giving fewer, but more forceful contractions. Ingestion of these plants can cause almost every type of arrhythmia, but bradyarrhythmias are the most common. Hyperkalemia may be noted. Gastrointestinal signs, seizures and sudden death may also be seen. Decontamination should include emesis if appropriate and repeated doses of activated charcoal due to enterohepatic recirculation. Avoid use of calcium containing fluids (LRS, Ringers). Monitor and correct arrhythmias as they arise (atropine, propranolol, lidocaine, phenytoin, etc.). Digibind® (digoxin immune Fab) is produced from specific digoxin antibodies from sheep. It will bind directly to the toxin and inactivate it. Unfortunately, it is expensive. The prognosis is guarded depending upon severity of clinical signs.

Kalanchoe sp. (devil's backbone, mother of millions, Mexican hat plant) is a common household plant, especially around the holidays. This plant contains a cardiac glycoside, but most dogs and cats only develop GI signs.

Grayanotoxin (andromedotoxin) containing plants include rhododendrons, azaleas (*Rhododendron spp.*), laurels (*Kalmia spp.*) and Japanese pieris (*Pieris spp.*). Grayanotoxins

bind to sodium channels in excitable cell membranes of nerve, heart, and skeletal muscle. They increase membrane permeability of sodium ions. Clinical signs include vomiting, hypersalivation, bradyarrhythmias, tremors, seizures and sinus arrest. Signs can last for 1 to 2 days. Emesis induction and activated charcoal can be given to asymptomatic animals. Atropine should be given for severe bradycardia.

Yews (*Taxus spp.*) are commonly used as landscaping plants. Yews contain taxine alkaloids which have a direct action on cardiac myocyte ion channels. They inhibit normal exchange of Na^+ and Ca^{++} across myocardial cells and depress cardiac depolarization leading to arrhythmias and fatal conduction disturbances. All parts, except for the ripe berry (fleshy red structure surrounding seed), are toxic. Sudden death can occur within hours of ingestion. Treatment includes minimizing stress and administering activated charcoal. Monogastrics that are still alive at 12 hours are likely to survive.

Tropane Alkaloid plants include Jimson weed (*Datura stramonium*), Belladonna (*Atropa belladonna*), and Henbane (*Hyoscyamus niger*). Clinical signs include tachycardia, dry mouth, mydriasis, hallucinations and possible seizures. The toxins are present in all parts of plant, but most concentrated in the seeds. If signs are life threatening, physostigmine can be given.

Solanaceous plants and solanine containing plants include: *Physalis spp.*, Ground cherry, *Cestrum spp.*, Jessamines and *Solanum spp.* (the nightshade group): potato, tomato, eggplant. Solanum alkaloids are severe GI irritants and can also contain cholinesterase inhibitors and variable amounts of atropine. Neurological effects from these plants can be mixed depending on the concentrations of solanine and atropine.

Tobacco (*Nicotiana spp.*) or cigarette ingestion is common in dogs. Tobacco contains nicotine and nornicotine (pyridine alkaloids). Nicotine mimics acetylcholine (ACh) at sympathetic and parasympathetic ganglia, at neuromuscular junctions of skeletal muscle, and at some synapses in the CNS (i.e., at nicotinic receptors). Vomiting is common, as nicotine affects the chemoreceptor trigger zone and emetic center. Low doses cause stimulation of receptors similar to ACh, while higher doses cause stimulation followed by blockage at autonomic ganglia and myoneural junctions of skeletal muscle (depolarizing blockade). Death is due to respiratory failure. Nicotine is poorly absorbed from the stomach, but well absorbed from the small intestine, respiratory tract, and skin. Alkalinization enhances absorption. The canine oral LD_{50} is 9.2 mg/kg. If the animal is asymptomatic, emesis should be induced followed by activated charcoal. Atropine can be given to treat the parasympathetic effects.

Marijuana (*Cannabis sativa*) is a very common ingestion in dog and cats. It is often co-ingested with other illicit substances or chocolate (brownies). The major active constituent in marijuana is THC (Δ -9-tetrahydro-cannabinol), although as many as 66 cannabinoids have been identified. The concentration of cannabinoids in plants depends upon environmental conditions. Signs can start within 30-60 minutes and last up to 72 hours. Most animals are

lethargic and ataxic, but about 25% are agitated instead. Treatment consists of supportive care. Rarely is marijuana intoxication lethal.

Hops (*Humulus lupulus*) are used in beer brewing. Ingestion of hops by dogs causes a malignant hyperthermia-like syndrome. Signs can be seen within three hours. Signs start with agitation, tachypnea, abdominal discomfort, and hyperthermia (104°F). There is a rapid progression to profound hyperthermia (> 108°F) and death. The toxic principle is unknown. If the animal is still asymptomatic induce vomiting and give a dose of activated charcoal. Start animal on intravenous fluids to prevent myoglobin or hemoglobin induced renal failure. If the dog is already hyperthermic institute cooling measures. Dantrolene (Dantrium®), a direct-acting skeletal muscle relaxant, works the best to reduce the temperature, but cyproheptadine can also be tried. Prognosis is poor to guarded, as many affected animals die.

Soluble Oxalate-Containing plants include: *Oxalis spp.* (shamrock), *Rheum rhaponticum* (rhubarb), *Rumex spp.* (dock, sour dock, curly dock), *Halogeton*, *Sarcobatus spp.* (greasewood) and *Chenopodium spp.* (lambsquarters). Soluble oxalates are sodium and potassium salts of oxalic acid. They are rapidly absorbed from the gut and hypocalcemia results from oxalic acid complexing with serum calcium. Fatal renal tubular necrosis may occur from crystallization of excreted calcium oxalate in the kidneys.

Lectins are plant proteins with an affinity for sugar moieties. They are also referred to as hemagglutinins and toxalbumins. Lectins are found in *Ricinus communis* (Castor bean) – toxic principle is ricin; *Abrus precatorius* (jequirity bean, rosary pea, precatory bean) – toxic principle is abrin; and *Robinia pseudoacacia* (black locust) – toxic principle is robin. Lectins disrupt protein synthesis in ribosomes leading to cell death. Rapidly dividing cells are the most severely affected. The lectins are found throughout the plant, but the highest levels are found in the seeds. The seed coat must be damaged to release the toxins. Clinical signs include lethargy, vomiting, diarrhea, abdominal pain, and hemorrhagic gastroenteritis. Treatment is symptomatic and supportive.

Autumn Crocus (*Colchicum autumnale*) contains colchicines which inhibits mitotic spindle formation. Ingestion can cause vomiting, diarrhea, weakness and possible death. Do not confuse with spring crocus (*Crocus spp.*) which is not toxic.