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An overview of potentially life-threatening poisonous plants in dogs and cats

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Abstract

Objectives: This article discusses the toxicity of the most common poisonous plants known to cause life-threatening systemic effects or death in dogs and cats as reported to the ASPCA Animal Poison Control Center (APCC).

Data sources: The article presents an overview of geographic distribution, toxic principle, clinical signs, clinical chemistry alterations, and treatment of intoxication as reported in the literature and based upon data retrieved from the APCC between January 2001 and December 2003.

Summary: Most plant exposures in dogs or cats result in mild to moderate signs of vomiting and diarrhea while liver, kidney, central nervous system, or cardiovascular effects are rare. Some garden or household plants can cause serious systemic effects or death when a small amount of plant material has been ingested. Based on APCC records, the most frequently reported poisonous plants causing serious systemic effects include Lily (*Lilium* and *Hemerocallis* spp.), Azalea (*Rhododendron* spp.), Oleander (*Nerium oleander),* Sago palm (*Cycas* spp.), Castor bean (*Ricinus communis*), Kalanchoe (*Kalanchoe* spp.), and Autumn Crocus (*Colchicum autumnale*).

Conclusion: Despite the variety of toxins present in the plants listed above, early clinical signs of toxicosis in dogs and cats can be nonspecific and can include vomiting, lethargy, anorexia, salivation, or diarrhea. Cardiac arrhythmias may be present with oleander, azalea, or kalanchoe ingestion. Evidence of liver or kidney damage (*Cycas, Lilium, Hemerocallis* spp., *Ricinus communis*) may occur 1–2 days after the exposure. Treatment consists of early decontamination and supportive care and may vary according to the type of plant involved and clinical signs present.

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Keywords: Autumn Crocus (*Colchicum autumnale*), Azalea (*Rhododendron* spp.), Castor bean (*Ricinus communis*), Kalanchoe (*Kalanchoe* spp.), lily (*Lilium* and *Hemerocallis* spp.), Oleander (*nerium oleander*), Sago palm (*Cycas* spp.), Toxicosis

Introduction

Dogs and cats can have access to several different poisonous plants in their environment. These plants may be available in the yard, garden, or brought into the house by the owner without knowing their toxic potentials. Another source of accidental exposure to poisonous plants is through a fresh flower bouquet containing lilies (Easter lily, stargazer lily). This often occurs on birthdays, Easter, Valentine's Day, Mother's Day, Father's Day, or on other festive occasions. Dogs or cats can often chew the leaves, stems, flowers, or occasionally seeds, and can develop a toxicosis. Dogs are likely to do this more often than cats because of their curious habits.

One of the common problems regarding plant poisoning exposures is correctly identifying the plant in question. Most plants present in the yard or in the household may have one or more common names. To confuse things further, common names for the same plant may vary from one region to another. In order to make an accurate diagnosis, correct identification of a plant is critical. Identification should only be made based on the scientific name of the plant, not on a common name. If the scientific name of the plant is not available, parts of the plant can be taken to a nearby florist, nursery, or to a botany department at a university or college for identification. Several Internet sites provide references and have good quality enlarged colored pictures of plants which can also aid in the identification.

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The toxicity of a plant can vary considerably depending on several factors such as the type of the plant involved, amount ingested, and sometimes the parts of the plant material ingested (some plant toxins are concentrated more in the seed or roots). Certain animal species may be sensitive to one type of plant and may show serious clinical effects while other species may be resistant to that specific plant.[1] Fortunately, most plant ingestions in dogs or cats result in only mild-to-moderate self-limiting signs of vomiting and diarrhea while systemic effects such as liver, kidney, central nervous system (CNS), or cardiovascular effects are rare. However, a few garden or household plants can cause serious systemic effects or death even when small amounts of plant material have been ingested. There have been several excellent articles and book chapters written on plant poisoning but most of this information pertains to large animals.[2–11]

The purpose of this article is to discuss the toxicity of the most frequently reported poisonous plants to the ASPCA Animal Poison Control Center (APCC) that can cause life-threatening systemic effects or death in dogs and cats. Based on APCC experience, a list of the most frequently involved poisonous plants causing serious systemic effects in dogs and cats was prepared. The list included Lily (*Lilium* and *Hemerocallis* spp.), Azalea (*Rhododendron* spp.), Oleander (*Nerium oleander*), Sago palm (*Cycas* spp.), Castor bean (*Ricinus comm*unis), Kalanchoe (*Kalanchoe* spp.), and Autumn Crocus (*Colchicum autumnale*). The article focuses on geographic distribution, toxic principles, clinical signs, clinical chemistry alterations, and management of these plant poisoning cases based on the veterinary and human literature and data retrieved from the APCC during January 2001 to December 2003.

Lilium and *Hemerocallis* (Day Lily) spp.

The APCC received 301 cases of *Lilium* species ingestion by dogs and cats during 2001–2003. *Lilium* species involved included Easter lily (*Lilium longiflorum*), tiger lily (*L. tigrinum*), rubrum lily (*L. speciosum*), stargazer lily (*L. auratum*), and Japanese show lily (*L. lancifolimu*). Most cases were reported in cats (83%). Preliminary assessment of these cases was based on exposure history and clinical information available, expected clinical signs of toxicosis described in the literature, amount ingested, and previous experience dealing with *Lilium* ingestion. APCC veterinarians believed 135 out of 301 cases (45%) had signs which were because of *Lilium* ingestion. Most frequently reported geographic location of cases was California (20%), New York (13%), Illinois (6%), Connecticut, New Jersey, Michigan, and Virginia (4%). The most commonly reported clinical signs were vomiting (31%), anorexia (9%), lethargy (9%), and anuria (4%). Acute renal failure was present in 3% of cases. Euthanasia was the result in 2% of cases. Fifty-nine cases of *Hemerocallis* species (day lily) exposure were reported to the APCC; most were in cats (62%). The APCC veterinary staff assessed 25 out of 59 cases (42%) related to day lily ingestion as intoxication. Most were from New York (23%), California (14%), Connecticut (14%), Virginia (11%) and Massachusettes (8%). The most commonly affected systems were gastrointestinal (28%), followed by CNS (6%).

Lilies considered toxic to cats are mainly found in the *Lilium* and *Hemerocallis* genera. Specifically identified species include Easter lily (*Lilium longiflorum*), tiger lily (*L. tigrinum*), rubrum lily (*L. speciosum*), stargazer lily (*L. auratum*,) (Figure 1), Japanese show lily (*L. lancifolium*), and some species of the day lily (*Hemerocallis* spp.) (Figure 2). It is important to know that neither lily-of-the-valley (*Convallaria majalis*) nor peace lily (*Spathiphyllum* spp.) belong to the *Lilium* or *Hemerocallis* genera, and thus, these are not considered true lilies. Lilies grow naturally along the Pacific Coast of the United States and in deciduous forests of the East, as well as on the Southern islands of Japan. Lilies are frequently cultivated as garden plants or houseplants.[2]

Lilies are known to cause acute renal failure in cats. Cats are usually exposed when these plants are present in the home, particularly during the Christmas or Easter holidays, as lilies are popular holiday ornamentals. These plants are also frequently used in flower bouquets. All parts of the plant are considered toxic, although the leaves are most frequently involved. In a recently published experimental study, nephrotoxicity was produced in cats by gavaging Easter lily plant extract. This study has shown that Easter lily flowers are much more toxic than the leaves. Although chemical identification of the toxin in Easter lily still remains unknown, it is believed that the toxin(s) is a water soluble compound.[12] Signs of lily toxicosis usually develop within 12 hours after exposure, although some reports indicate onset of these signs from 2 hours to 5 days after exposure.[13–15] Illness progresses to kidney failure in 2–3 days.[13] In day lily (*Hemerocallis*) toxicosis in cats, the incidence of acute renal failure after exposure was 32%.[16] Clinical signs of kidney failure include polyuria, oliguria or anuria, dehydration, vomiting, diarrhea, and depression. In a case report of 6 cats exposed to day lily, 2 were anuric, 2 were polyuric, and 1 cat was oliguric.[14] Anuria and oliguria were the most common findings compiled by the APCC in previous reports.[13] Some cats are presented with CNS signs such as ataxia, head pressing, disorientation, tremors, and seizures.[1,16]

Serum chemistry findings in lily toxicosis are reflective of renal failure and include increased blood urea

nitrogen, creatinine, phosphorus, and potassium. Azotemia is usually severe, but creatinine may be disproportionately elevated.[13] Epithelial casts usually develop and can be seen in the urine starting 12 hours after exposure. Urinalysis also typically reveals glucosuria.[13] Histopathologic examination of the kidneys shows renal tubular necrosis with or without mineralization.

Treatment of lily toxicosis consists primarily of supportive care. The objectives of treatment are aimed at early decontamination, prevention of renal failure, and maintenance of fluid, electrolyte, and acid–base balance. Renal damage can be minimized in cats that have been decontaminated and have had early diuresis.[13,15] A delay in treatment 18 hours or longer after ingestion generally results in renal failure.[13] If exposure has taken place within 2 hours of clinical presentation and no clinical signs are present, induce emesis in cats with 3% hydrogen peroxide at 1.5 mL/kg PO. This should be followed by administration of activated charcoal at 1–2 mg/kg along with a cathartic such as sorbitol (70.0%) at 1–3 mL/kg or magnesium sulfate or sodium sulfate at 250 mg/kg PO. Intravenous (IV) fluid therapy is required for at least 48 hours to induce diuresis.[1,13,14] Urine output should be monitored. Severe intoxications may require hemodialysis, peritoneal dialysis or kidney transplant.

Rhododendron species (Azaleas)

The APCC received 188 cases of azalea ingestion in dogs and cats; 98 of these cases had signs which were consistent with azalea ingestion. Most were reported from California (13%), Virginia (10%), Connecticut (8%), Illinois (8%) and Massachusetts (8%). The most common signs were vomiting (28%), depression (10%), diarrhea (8%), and anorexia (5%).

Azaleas grow naturally in the Eastern and Western United States mountain woods as a wild flowering plant throughout the spring and summer (Figure 3). Distribution extends from cool-temperate areas of the Northern hemisphere into the Southern hemisphere and Southeast Asia. Azaleas are also displayed as ornamental plants in households.[17] The toxic principles are grayantoxin glycosides. Grayantoxins are diterpinoid compounds.[3] Grayantoxins (also known as andromedotoxin, acetylandromedol, rhodotoxin) have been shown to bind to and produce a modified opening of sodium channels which delays repolarization of ventricular muscle fibers in dogs.[1,18] Such continued depolarization excites cell membranes. Various derivatives of grayantoxin are present in all portions of the plant, including the nectar.[1]

The toxin manifests its effect in the heart as either tachycardia or bradycardia. Gastrointestinal signs such as ptyalism, vomiting, bruxism, diarrhea, or constipation may also develop. Cardiovascular changes may lead to weakness, hypotension, difficulty breathing, CNS depression, coma, collapse, convulsions, and death.[1,18,19]

Serum chemistry changes noted with rhododendron intoxication are usually non-specific. Chromatographic methods are available to determine the presence of grayanotoxins in serum, urine, and stomach contents. Identification of grayanotoxins in body fluids may help when making a diagnosis.[4] Unfortunately, diagnostic results based on plant toxin identification will not be available quickly enough to affect initial case management.

Initial treatment of azalea toxicosis includes induction of emesis provided vomiting has not already occurred. Induce emesis with 3% hydrogen peroxide at 1.5 mL/kg PO in dogs or cats or with apomorphine in dogs at 0.02–0.04 mg/kg conjunctivally or IV. Emesis should be followed by administration of activated charcoal. Heart rhythm should be monitored with an electrocardiogram (ECG), and blood pressure measurements obtained at regular intervals. Atropine should be administered (0.02–0.04 mg/kg IV) if bradycardia is present. Isotonic fluids should be given to correct hypotension. Catecholamines, such as dopamine or norepinephrine, may be needed if blood pressure is not normalizing with fluid therapy alone.[1]

Nerium oleander (Oleander)

Seventy-one cases of oleander ingestion in dogs and cats were reported to the APCC; 42 of these cases had signs which were consistent with oleander ingestion. Most were reported from California (34%), Arizona (13%), Texas (11%), and Florida (8%). The most common signs were vomiting (26%), depression (8%), diarrhea (7%), and cardiac arrhythmia (4%).

Oleander, native to Asia and the Mediterranean, has been naturalized outdoors in warmer regions of North America (Figure 4). It may be found inside homes as an ornamental plant. It can also grow as a shrub, so it may be used in yards for hedging.[19–21] Dogs and cats are exposed by ingesting fresh or dried parts of the plant. Although the plant is reportedly unpalatable, hungry, or bored animals will eat them. The toxic principles are steroidal glycosidic cardenolides, a type of cardiac glycoside. Structurally, cardenolides are steroids with 23 carbons and at least 1 sugar group attached to the C-3 position.[22] Many cardenolides have been identified in different concentrations in different parts of the plant. However, all parts have enough concentration of the toxins to cause clinical signs. The oleander glycosides believed responsible for toxicity include oleandrin, neriine, and some other glucosides.[21] These compounds are structurally similar to digitalis and inhibit the so-

Figure 1: Stargazer Lily Bouquet.

Figure 2: Day Lily (*Hemerocallis* sp.).

Figure 3: Azalea (*Rhododendron* sp.).

Figure 4: Oleander (*Nerium oleander*).

dium-potassium ATPase by inducing a conformational change in the enzyme, preventing active transport of sodium, thereby allowing potassium out of the cell.[23]

Clinical signs of oleander toxicosis include vomiting and diarrhea with or without blood, CNS depression, and hypersalivation. Cardiac abnormalities may include bradycardia, tachycardia, AV block, or various arrhythmias because of increased sympathetic tone, leading to sinus arrest. Increased sympathetic tone also may cause mydriasis in animals after oleander ingestion.[19–21] Additionally, cold extremities, hypotension, pale mucous membranes, weak and irregular pulse, muscle tremors, collapse, and coma may develop because of decreased cardiac output. Interestingly, males tend to be more affected with clinical signs than females post-exposure. The onset and duration of clinical signs are dependent upon the amount of toxin ingested and treatment administered. Signs may develop within 45 minutes and last over 4–5 days, ending in either death or recovery. Serum chemistry may reveal hyperkalemia.[20,23] Presence of oleandrin in the stomach contents or other body fluids will confirm exposure.[24]

Treatment of oleander toxicosis in dogs and cats consists of early decontamination (induction of emesis and charcoal administration), and symptomatic and supportive care. Repeated doses of activated charcoal may prevent enterohepatic recycling of the toxins.[20] Cardiac rhythm should be monitored with an ECG for at least the first 24 hours. Digoxin- specific antibody fragments (Digibind™)[a] help to reverse cardiotoxic effects of the glycosides.[20] Potassium chloride can be given to treat hypokalemia. Cardiac arrhythmias can be treated with atropine or propanolol as needed.[19–21] It has also been demonstrated experimentally that fructose-1,6-diphosphate prevents the severity of cardiac and ion imbalance effects in dogs, although the mechanism behind this action is unknown.[23]

Cycas species (Sago palm)

The APCC received 69 calls concerning Sago palm ingestion in dogs and cats during 2001–2003; most were reported in dogs (99%). On the basis of exposure history and clinical signs present, 60 out of 69 cases (87%) were because of Sago palm ingestion. Most cases were reported from Texas (36%), Florida (34%), California (10%), Louisiana (5%), and North Carolina (2%). Most commonly reported clinical signs were vomiting (35%), lethargy (14%), diarrhea (6%), and anorexia (5%). Death was reported in 3% of the cases.

Sago palms or cycad palms are distributed natively across the tropics, subtropics, southern United States, Hawaii, or are brought into homes as ornamental plants (Figure 5). These plants are frequently used as part of a landscaping scheme. In dogs, most exposures occur in the southern United States.[25–27] All parts of the plant contain toxins, but seeds contain the highest concentrations. As little as 1–2 seeds can be lethal to an average size dog. The toxins believed to be responsible for toxicosis are the azoglycosides cycasin and methylazomethanol, a neurotoxic amino acid (β-*N*-methylamino-L-alanine), and an unidentified high molecular weight compound. The glucose molecule on cycasin is hydrolyzed by the gut bacterial enzyme β-glycosidase, yielding sugars and methylazoxymethanol. This compound then alkylates DNA and RNA, causing hepatotoxic, teratogenic, carcinogenic and gastrointestinal effects. The toxins methylazomethanol and β-*N*-methylamino-L-alanine are unlikely to be the cause of severe neurologic damage seen in cattle since it has been proposed that cattle can extensively metabolize and inactivate these toxins in the rumen.[27] An unidentified high molecular weight compound may be responsible for this neurologic damage.[25–27]

Clinical signs typically begin with vomiting and diarrhea (with or without blood). Other signs can include constipation, hypersalivation, abdominal tenderness, and anorexia.[1,26] Clinical signs are generally observed within 24 hours after ingestion. Evidence of liver damage is generally delayed for 2–3 days. Neurologic signs include weakness, ataxia, and proprioceptive difficulties. In severe cases, seizures and coma have been noted. Reports in dogs have also included evidence of ascites, as well as ecchymotic hemorrhage (noticed both ante and post-mortem).[1,25–27] Mortality can be up to 33% in dogs.[25]

Changes in serum chemistry in sago palm toxicosis indicate liver damage: elevations in conjugated bilirubin, alanine aminotransferase, alkaline phosphatase, white blood cell count, and decreased total protein and platelets.[1,25–27] Increases in prothrombin time and partial prothromboblastin time can be observed. Thrombocytopenia may occur in conjunction with coagulopathy.[1,26,27] Azotemia may develop secondary to hepatic damage, and decreased renal blood flow because of hypotension. Urinalysis results can show glucosuria, bilirubinuria, and hematuria.

Objectives of treatment of sago palm toxicosis are early decontamination, monitoring liver function, controlling CNS and GI signs, and supportive care. Induction of emesis followed by administration of activated charcoal may limit the amount of toxin absorbed. Fluid therapy and GI protectants, such as cimetidine (5–10 mg/kg orally 3–4 times per day) or sucralfate (0.5–1 g/dog, or 125–250 mg/cat) orally every 8–12 hours should be administered if evidence of gastric ulceration is present.[25] Intoxicated animals should be monitored and treated as needed for secondary effects of acute hepatic failure such as hepatic encephalopathy, coa-

gulopathy, and hypoproteinemia (see castor bean for further treatment recommendations). Vitamin K_1 administration and blood transfusions with fresh whole blood should be considered in cases of massive hemorrhage or anemia. Monitor liver enzymes on presentation and at 24, 48, and 72 hours post-exposure or until resolution of clinical signs. Diazepam or other anticonvulsants are helpful to control seizures.[1,25]

***Ricinus communis* (Castor bean)**

Thirty-five cases of castor bean ingestion in dogs were reported to the APCC during 2001–2003; none were reported in cats. On the basis of exposure history and clinical information available, 21 out of 35 cases (60%) had clinical signs that were considered because of castor bean ingestion. Most cases were reported from Arizona (10%), Ontario, Canada (10%), Virginia (10%), District of Columbia (8%), Indiana (8%), and New Jersey (8%). The most common clinical signs were vomiting with or without blood (38%), lethargy (14%), anorexia (12%), depression (8%), diarrhea (6%), and neurologic signs including weakness, tremors, and seizures (2%).

Castor bean plants grow in the Tropics, Africa, West Indies, and also as a decorative or ornamental garden plant in the US. Castor plants have been naturalized in the Southern US where they thrive in warmer weather (Figure 6). The beans are commercially grown for castor oil.[28,29] The plant and beans are used for ornamental purposes, including pieces of jewelry.[1]

The toxic principle is ricin, a heterodimeric glycoprotein, which is a toxalbumin. Toxalbumins act to inhibit protein synthesis. The protein is composed of an A and B subunit linked by one disulfide bond.[28] The B glycoprotein chain has a high affinity for mammalian cell membranes, inducing endocytosis following glycoprotein binding. Once inside the cell, the α chain of the ricin molecule enters the endoplasmic reticulum and depurinates the 28S ribosomal subunits. This is an effective mechanism for inducing cellular necrosis.[1,28,29] All parts of the plant contain ricin; the highest concentration is found in the bean.[28] Therefore, ingestion of the bean is considered more toxic than the other parts of the plant.

Clinical signs of toxicosis in dogs may develop within 6 hours of ingestion or may be delayed up to 24 hours.[1,28] The severity of signs increases if the seed is chewed open, allowing the ricin to be released quickly. Ricin is not likely to be released from the seed unless the coat is broken or damaged.[28] Most common signs include vomiting, severe diarrhea (which may be watery or bloody), depression, nausea, abdominal pain, and anorexia. Additional signs may include tenesmus, dehydration, polydipsia, hypersalivation, weakness, muscle twitching, tremors, seizures, coma, and death.[1,28,29] The development of clinical signs depends on the route of administration of the toxin. In an experimental report in dogs, ricin was given IV and animals developed weakness, anorexia, apathy, and moderate fever. Mortality occurred 15–40 hours after lethal doses were given. No microscopic evidence of kidney or liver damage was observed, despite previous experimental reports of ricin-induced liver and kidney necrosis.[30] In fatal non-experimental cases, seizures may precede death, which generally results from hemorrhage in the heart and lungs. Clinical signs can last up to 5 days.[28,29] Mortality in dogs has been reported up to 9%.[28]

Changes in serum chemistry may include elevated alanine aminotransferase, aspartate aminotransferase, and lactate dehydrogenase, indicative of liver damage.[28] Increased blood urea nitrogen and serum creatinine may indicate the presence of kidney damage. Additionally, an increase in the white blood cell count is typical. Increased serum albumin and globulin may also be seen.[1,29]

Treatment is supportive and symptomatic. Liver and kidney function, along with electrolytes, should be monitored on presentation, at 24 and 48 hours, or until resolution of all clinical signs. Induce emesis with a recent exposure, followed by administration of activated charcoal. Administer GI protectants such as sucralfate and H_2 blockers (cimetidine, famotidine, ranitidine) and IV fluid therapy for 2–3 days or as long as needed. Administer diazepam for seizures. If liver damage has already occurred, oral antibiotics and lactulose (15–30 mL PO every 6–8 hours in dogs, and 0.25–1 mL PO every 8–12 hours in cats) may be used in the management of hepatic encephalopathy. Other therapies may include administration of SAM-e[b] (18 mg/kg orally in dogs and cats every other day to every 3rd day for 1–3 months), and dietary management.[1,28,29]

***Kalanchoe* spp.**

The APCC received 30 calls concerning possible Kalanchoe exposure of which 12 had clinical signs which were considered because of Kalanchoe ingestion (4 were in dogs and 8 in cats). Most cases were reported from Illinois (33%) followed by California (17%), New York (17%), and Pennsylvania (17%). The most common signs were vomiting (23%), depression (13%), tachycardia (7%), and diarrhea (7%).

Kalanchoes grow in South Africa, South Arabia, along the coast of India, Sri Lanka, Madagascar, and on other islands in the Indian Ocean. Kalanchoe has been naturalized in Australia, South and North America. In North America, this plant is normally an indoor houseplant (Figure 7) and may be seen especially during

Figure 5: Sago Palm Cones and Leaves.

holidays.[31] The toxic principles are cardiotoxic bufadienolides, which are cardiac glycosides. Cardiac glycosides inhibit the sodium-potassium ATPase in myocytes, thus allowing potassium to leak out of the cell, and keeping calcium and sodium trapped in the cell.[22] Three bufadienolides have been isolated from Kalanchoe, one of which is hellebrigenin 3-acetate. The identities of the other 2 bufadienolides remain unknown.[22] The toxic compounds are located in all parts of the plant, with the highest concentration in the flowers. Different species of this genus have varying levels of toxicity, although all are considered toxic.[31,5]

The toxins affect the GI, cardiovascular, and neuromuscular systems. Acute clinical signs include hypersalivation, polyuria, depression, diarrhea, and inappetance. Cardiac and respiratory abnormalities may develop within 1–2 days. Cardiac abnormalities such as AV block, and bradycardia may be present. Severe

Figure 6: Castor Bean Leaves and Seed Pods.

Figure 7: Kalanchoe Flowers and Leaves.

Figure 8: Autumn Crocus Leaves and Flower (courtesy Dr. Erick Dunayer).

weakness and paresis of the neck region, followed by ataxia and paralysis, have been seen experimentally in guinea pigs and sheep.[6] Weakness can progress to collapse and death within 4–5 days after exposure. In severe cases, death may occur within hours.[31] In cattle, myocardial edema, epicardial, pulmonary, and GI hemorrhage were noted at necropsy. Cyanosis is also another consistent finding among guinea pigs, sheep, and cattle.[7]

Changes in serum chemistry may include increased blood urea nitrogen and creatinine secondary to mild renal disease or dehydration. Increased blood glucose may also be observed.[30] There may be an increase in blood carbon dioxide levels.

Treatment recommendations have mostly been described in the literature for bovine intoxications because this is the species most often affected. Early decontamination is warranted with induction of emesis and repeated doses of activated charcoal. Depending on the cardiac abnormality seen, atropine or a β-blocker such as propanolol should be administered (see azaleas and oleander for further treatment recommendations). In cattle, treatment is usually effective within 24 hours of intoxication.[31,7]

***Colchicum autumnale* (Autumn Crocus)**

The APCC received only 3 calls concerning *Colchicum* ingestion in dogs and cats; 2 of these had clinical signs which were believed to be because of *Colchicum* ingestion. Cases were reported from Ontario, Canada, and Maryland. Vomiting and lethargy were the most commonly reported clinical signs.

Colchicum is naturally distributed across Europe and Asia, but is rarely seen in North America (Figure 8) except as an ornamental plant.[8,9] Pets are likely to be exposed when this plant is brought into a household. The toxic principles include mainly colchicines, but other alkaloids are involved as well. Colchicine is an alkaloidal amine derived from phenylalanine.[10] Unlike most alkaloids, it does not have a heterocyclic nitrogen among the structure.[11] Colchicine arrests mitosis in living cells, probably at the metaphase stage, resulting in the disruption of microtubules. Microtubule disruption prevents formation of the spindle apparatus in prometaphase, leading to a non-viable cell. Microtubule formation also produces abnormal nuclear formations that lead to apoptosis.[9,32] Originally used to treat gout, this alkaloid is currently used as a treatment for inflammatory diseases, and has been classified as a microtubule-disrupting drug for these purposes.[10,32]

The toxin results in clinical signs of inappetance, vomiting, diarrhea, hypersalivation, depression, and abdominal pain progressing to hemorrhagic gastroenteritis. Mitotic arrest in the intestine causes the crypt epithelium to swell, resulting in malabsorption. Other signs may include weakness, incoordination, paresis or collapse. Kidney failure has also been reported.[8,10] In experimentally-induced acute toxicoses in cats, colchicine caused severe CNS depression, hypothermia, proprioceptive deficits, coma, convulsions, respiratory arrest, and death. When cats were given colchicine on a chronic lower-dose basis, signs included mild anorexia, lethargy, and severe hindlimb atrophy after 3 weeks.[33]

Serum chemistry and hematology changes may include increased blood urea nitrogen, erythrocytosis, and leukopenia.[9] Histopathologic findings indicate widespread tissue necrosis with karyopyknotic and karyorrhectic nuclei. Mitotically arrested cells are seen in the GI epithelium, lymphoid, and hemopoietic systems.[32] The morphologic pathology of these cells from all 3 systems correlates well with higher concentrations of alkaloid within the bone marrow compared with other tissues.[34]

Treatment of colchicine toxicosis consists of early decontamination and supportive care. Administration of fluid therapy and GI protectants, such as sucralfate, is necessary. Monitor for myelosuppression, anemia, and leukopenia. Use of erythropoietin or filgastrim[c] may be helpful for these conditions.

Conclusion

Although the plants described above have varied geographic distributions and some are not native to the United States, many of them are frequently present in yards, gardens or households in the United States. During the study period, the most serious plant poisoning cases received by the APCC involved *Lilium* spp., *Hemerocallis* spp., *Rhododendron* spp., *Nerium oleander, and Cycas* spp, *Kalanchoe* spp.), and *Colchicum autumnale*. Despite the variety of toxins present in these plants, early clinical signs of toxicosis in dogs and cats can be non-specific and can include vomiting, lethargy, anorexia, salivation, or diarrhea. Cardiac arrhythmias may be present with oleander, azalea, or kalanchoe ingestion. Evidence of liver or kidney damage (*Cycas, Lilium, Hemerocallis* spp., *Ricinus communis*) may occur 1–2 days after the exposure. Most cases are reported from the Southwest, Eastern or Midwest United States. Obtaining an accurate history and positive identification of the plant in question is a critical part of determining a diagnosis and effective treatment. Treatment is primarily symptomatic and supportive and may vary according to the type of plant involved and clinical signs present. After early decontamination, implement fluid therapy, seizure control and monitoring cardiac, liver, or kidney functions. Monitoring complete blood

count and serum chemistries on presentation and 24, 48, and 72 hours after the exposure may be necessary.

Footnotes

[a] Digibind®, Digoxin Immune Fab (Ovine) Specific Antibody for Digoxin, Glaxo Wellcome.
[b] Denosyl®, S-Adenosyl-Methionine (SAMe).
[c] Neupogen®, Filgrastim, Amgen.

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