

PEER-REVIEWED

Spring-blooming bulbs: A year-round problem

Camilla L. Lieske, DVM, MPVM

Bulbous flowering plants are popular for beautifying the home and garden, but these plants or their bulbs can pose a toxic threat to pets. In addition to the bulbs themselves potentially acting as foreign body obstructions (especially if they are ingested whole), compounds within the bulbs, leaves, flowers, or stems can have a variety of toxic effects on animals. Dogs may be more likely to ingest the plants than are other pets. Although most of these plants grow and flower during the spring, they present a year-round toxic threat: in the autumn during the planting season, in the spring while they are growing, or anytime bulbs are grown in pots inside the home.

The term *bulb* technically includes only plants with underground stems surrounded by modified leaves called *scales*, which store nutrients. However, gardeners commonly apply the term to any plant with a bulbous root (or underground stem), be it a corm, tuber, rhizome, or true bulb.

This article discusses the effects of some of the more common spring-blooming bulbous plants. Although most of these plants belong to the Iridaceae and Liliaceae families, their toxic effects vary. These effects are summarized in *Table 1*. The plants are sorted and discussed based on their clinical effects: contact dermatitis, mild to moderate gastrointestinal (GI) irritation, moderate to severe GI irritation or damage, severe multisystemic signs, and cardiotoxic signs. Plants with more than one effect are discussed in multiple categories.

Contact dermatitis

In addition to GI signs resulting from ingestion, several bulbous plants are associated with contact dermatitis.

“Toxicology Brief” was contributed by Camilla L. Lieske, DVM, MPVM, Department of Veterinary Biosciences, College of Veterinary Medicine, University of Illinois, Urbana, IL 61802. The department editor is Petra A. Volmer, DVM, MS, DABVT, College of Veterinary Medicine, University of Illinois, Urbana, IL 61802. Dr. Volmer is also a diplomate of the American Board of Toxicology.

Examples of plants in this category are *Hyacinthus* (hyacinth; *Figures 1A & 1B*), *Narcissus* (daffodil, jonquil, narcissus; *Figures 2 & 3*), and *Tulipa* (tulip) species. Dermatitis has been reported in people working in occupations associated with the flower industry, especially in flower pickers.^{1,2} There is evidence that the effect in *Tulipa* and *Hyacinthus* species is due to contact allergens found in the sap of the plants (tuliposide A in *Tulipa* species, an unknown compound in *Hyacinthus* species).² Another mechanism that contributes to the dermatitis seen with *Hyacinthus* species is mechanical irritation due to calcium oxalate raphides (needlelike crystals).³

Dermatitis caused by *Narcissus* species also appears to be an irritant condition resulting from exposure to calcium oxalate raphides found in the plant's sap. Each species of *Narcissus* has a different severity of effect. *Narcissus pseudonarcissus* (daffodil) has the most severe effects, while *Narcissus tazetta* (paper white) rarely results in problems.¹ Although no dermatitis cases in animals have been reported for these plants, there have been reported cases of dermatitis with amaryllis, a related plant.⁴

Clinical signs of contact dermatitis generally resolve by eliminating exposure. Bathing in a mild shampoo and using humectants may be beneficial in severe cases.

Mild to moderate GI irritation

Plants that can cause mild to moderate GI signs include *Crocus* species (crocus; *Figure 4*), *Galanthus nivalis* (snowdrop), *Hyacinthus* species, *Muscari armeniacum* (grape hyacinth; *Figure 3*), and *Tulipa* species. Although severe signs are not expected, ingesting large amounts of plant or bulb material from these species may result in GI irritation.

Galanthus species contain several alkaloids, including lycorine found also in *Narcissus* species (see *below*), located mostly in the bulb.³ *Tulipa* species contain compounds that may act as contact irritants or allergens, including tulipalin A and B and tuliposide A, concentrated in the bulb.^{2,3} The toxic agents in *Hyacinthus* species, *M. armeniacum*, and *Crocus*

Continued



FIGURE 1A



FIGURE 1B



FIGURE 2

1A & 1B. These blue (1A) and white (1B) hyacinths can cause contact dermatitis and mild or moderate GI signs.

2. This cat is close to two toxic plants. The daffodils on the right can cause contact dermatitis and moderate or severe GI signs. The lilies of the valley on the left can be cardiotoxic.

3. Two toxic plants in one arrangement. The yellow daffodils in the center can cause contact dermatitis and moderate or severe GI signs. The grape hyacinths on the edges can cause mild or moderate GI signs.

4. This crocus plant can cause mild or moderate GI signs if ingested.



FIGURE 3



FIGURE 4

species have not been well-described.

Clinical signs in animals include nausea, vomiting, and diarrhea (ASPCA Animal Poison Control Center [APCC] Database: Unpublished data, 2002).³ Unless an animal ingests large amounts, treatment at a veterinary clinic is usually not needed. Activated charcoal (1 to 4 g/kg orally in 50 to 200 ml water⁵) may be beneficial in cases of recent ingestions. To prevent vomiting that often occurs with large initial doses, start at the low end of the dose and repeat it. Treatment is otherwise symptomatic: antiemetics (metoclopramide hydrochloride 0.2 to 0.4 mg/kg t.i.d. to q.i.d. orally, subcutaneously, or intramuscularly, or 0.01 to 0.02 mg/kg/hr as a continuous intravenous infusion)⁶ and GI tract protectants (attapulgit [Kaopectate] 1 to 2 ml/kg orally every four to six hours or sucralfate 0.5 to 1 g [dogs], 0.25 to 0.5 g [cats] orally b.i.d. to t.i.d.).⁶ Also monitor animals for dehydration if protracted vomiting and diarrhea develop.

Keep in mind that *crocus* is commonly used to refer to two genera of plants, *Crocus* and *Colchicum*.

Although the most common *Crocus* species are spring-blooming, there are *Crocus* species that bloom in the autumn, such as *Crocus sativus* (saffron) and *Crocus speciosus*.⁷ It is important not to confuse the *Crocus* species with the *Colchicum* species, both commonly called *autumn crocus*. *Crocus* species ingestions generally will not cause more than mild GI signs; however, ingestions of *Colchicum* species may result in death (*see below*). *Crocus* and *Colchicum* species may be distinguished by the size of the plant. Although there are some exceptions, *Crocus* species generally do not grow larger than 6 in tall, while *Colchicum* species are most commonly 8 to 12 in tall.⁷ Because of the potential lethality of *Colchicum* species ingestion, assume all autumn-blooming crocus ingestions are *Colchicum* species, unless there is certainty that the ingestion was *Crocus* species.

Moderate to severe GI irritation or damage

Plants that can cause moderate to severe GI signs include *Cyclamen* species (cyclamen), *Gladiolus*

TABLE 1 Bulbous Flowering Plants and Their Clinical Adverse Effects*

Scientific Name	Common Name	Family	Adverse Effects	Toxic Agent
<i>Arisaema triphyllum</i>	Jack-in-the-pulpit	Araceae	Oral swelling, hypersalivation, gastritis, GI ulceration, dyspnea	Calcium oxalate raphides
<i>Colchicum autumnale</i>	Autumn crocus, meadow saffron	Liliaceae	Vomiting, hemorrhagic diarrhea, ataxia, paresis, liver and kidney failure, death	Colchicine
<i>Convallaria majalis</i>	Lily of the valley	Liliaceae	Vomiting, diarrhea (may be hemorrhagic), cardiac arrhythmias, seizures, death	Cardenolides
<i>Crocus</i> species	Crocus, saffron	Iridaceae	Vomiting, diarrhea	Unknown
<i>Cyclamen</i> species	Cyclamen	Primulaceae	Vomiting, diarrhea (may be hemorrhagic); possible cardiac arrhythmias and seizures	Saponins (glycosides)
<i>Galanthus nivalis</i>	Snowdrop	Liliaceae	Vomiting, diarrhea	Alkaloids
<i>Gladiolus</i> species	Gladiola	Iridaceae	Hypersalivation, severe gastritis, GI ulceration	Unknown
<i>Gloriosa superba</i>	Glory lily	Liliaceae	Vomiting, hemorrhagic diarrhea, ataxia, paresis, liver and kidney failure, death	Colchicine
<i>Hyacinthus</i> species	Hyacinth	Liliaceae	Contact dermatitis, vomiting, diarrhea	Unknown
<i>Iris</i> species	Iris	Iridaceae	Hypersalivation, severe gastritis, GI ulceration, death	Irisin, terpenoids
<i>Muscari armeniacum</i>	Grape hyacinth	Liliaceae	Vomiting, diarrhea	Unknown
<i>Narcissus</i> species	Daffodil, jonquil, narcissus	Liliaceae	Possible contact dermatitis, severe gastritis, GI ulceration, seizures, hypothermia, death	Calcium oxalate raphides, alkaloids (lycorine)
<i>Tulipa</i> species	Tulip	Liliaceae	Contact dermatitis, vomiting, diarrhea	Tuliposide A, tulipalin A and B

*Most are spring-blooming plants with a few exceptions: *Gladiolus* species are summer-blooming bulbs, but have the same adverse effects as *Iris* species; *Colchicum* species are autumn-blooming bulbs, commonly confused with *Crocus* species; *Gloriosa superba* are summer-blooming bulbs with the same adverse effects as *Colchicum* species.

Continued

species (*gladiola*), *Iris* species (iris), *Narcissus* species, and *Arisaema triphyllum* (jack-in-the-pulpit). Ingestion of any of these plants is likely to result in GI signs, although the toxic principles for each plant type are not all the same. The severity of signs depends on the amount of the plant (or portion of the plant) consumed.

Cyclamen species contain several irritating saponins (soapylike plant glycosides), including cyclamin, isocyclamin, cyclaminorin, and cyclacoumine.³

Do not confuse *Crocus* species with *Colchicum* species, both commonly called *autumn crocus*.

Although clinical signs are usually limited to a purgative effect (vomiting and diarrhea), some of the saponins (*e.g.* cyclamin) may have cardiac effects similar to cardiac glycosides. Cardiac arrhythmias and seizures are possible with large ingestions. Hemorrhagic diarrhea may also be seen. It has not been determined how much of the plant needs to be ingested to see signs more severe than the GI effects. Treatment is symptomatic; most animals will recover without treatment.³

The clinical signs produced by *Gladiolus* and *Iris* species ingestions are similar, although only *Iris* species have a well-elaborated toxic principle. Rhizomatous *Iris* species are toxic in all animal species.³ Toxicosis is associated with ingestion of a small quantity of the bulbs or rhizomes or a large quantity of the leaves. Several toxic compounds have been identified, including the resinous purgative irisin (considered the primary toxicant) and cytotoxic terpenoids and quinines; the contribution of the cytotoxic compounds to any toxicosis is undetermined.³ The toxicant in *Gladiolus* species is not well-described, although it appears to be more concentrated in the corms than in the leaves. Clinical signs of *Gladiolus* or *Iris* species exposure include salivation, vomiting, diarrhea, anorexia, and ulcers and hemorrhage of the

stomach and small intestine (ASPCA APCC Database: Unpublished data, 2002).³ In people, *Iris* species ingestions have resulted in a burning sensation in the mouth and hypersalivation.³ A case of *Iris* species ingestion in calves resulted in hypersalivation, oral ulcers, gastritis, and death.⁸ In cases of bulb, corm, or rhizome ingestion or ingestion of a large amount of leaves, decontamination with emesis (when appropriate) and activated charcoal is recommended. Direct symptomatic treatment toward alleviating signs and protecting the GI tract (*e.g.* administering metoclopramide, sucralfate, and H₂ antagonists, as needed). Monitor and treat for dehydration as appropriate.

The primary suspected toxins in *Narcissus* species are alkaloids. Most of the alkaloids isolated from *Narcissus* species are found in the bulbs, although alkaloids are also present in leaves and flowers. *Narcissus* species contain many alkaloids; the primary one is lycorine (also called narcissine).^{3,9} Other alkaloids include galanthamine hydrobromide (central and peripheral inhibition of acetylcholinesterase) and coccinine (convulsant in high dosages),³ although the amount of plant that needs to be ingested to see signs associated with these alkaloids has not been determined. Calcium oxalate raphides may also play a role in the clinical signs by causing mechanical damage to the gastrointestinal mucosa. Human exposures often result from mistaking *Narcissus* species bulbs for onions or shallots.¹⁰ Clinical signs are usually consistent with lycorine exposure and consist of vomiting and diarrhea (ASPCA APCC Database: Unpublished data, 2002). Hypothermia has also been reported (ASPCA APCC Database: Unpublished data, 2002).⁹ Severe gastritis and GI ulceration can result (ASPCA APCC Database: Unpublished data, 2002).³ Trembling, convulsions, and death have been reported in human ingestions.⁹ Death has also been reported in cattle after ingestion of *Narcissus* species.⁹ Animals prone to seizures are more likely to experience severe seizures, and vomiting may trigger their

Continued

seizures. As with *Gladiolus* and *Iris* species, treatment involves decontaminating an asymptomatic animal. Direct symptomatic treatment toward ameliorating signs. Anticonvulsants (e.g. diazepam or a barbiturate such as phenobarbital if diazepam is ineffective) may be needed.¹¹ Signs generally last from hours to a day.³

The toxic principle in *A. triphyllum* is the same as that in other members of the Araceae family (commonly known as the arum family), including the genera *Philodendron* and *Dieffenbachia*. Clinical signs are the result of mechanical damage to the GI mucosa by insoluble calcium oxalate raphides. In addition to the mechanical damage, chemical compounds found in the plant may result in the release of inflammatory mediators, including histamine and prostaglandins. The characteristic grooves along the raphides in this family may channel the chemical agents into the wounds.^{3,12} Signs of exposure include redness and swelling of the oral mucosa, pain, hypersalivation, vomiting, and diarrhea. If the oral and esophageal swelling is severe enough, animals may become dyspneic. Initial treatment should include thoroughly rinsing the oral cavity. Monitor animals for dehydration, electrolyte imbalances, and shock, and treat them as necessary. Symptomatic treatment revolves around reducing pain and inflammation. Antihistamines, such as diphenhydramine, may be effective,¹² although some reports indicate they are of limited value.³ To relieve pain and swelling in people, lime juice is reported to be more effective than other treatments, such as milk, water, or ice.¹³

Severe multisystemic signs

Both *Colchicum autumnale* (autumn crocus, meadow saffron) and *Gloriosa superba* (glory lily) contain colchicine. Colchicine is concentrated in the corm and seeds of *C. autumnale*, although all parts of the plant are toxic. The lethal dose is estimated at 8 to 16 g fresh leaves/kg in cattle, 6.4 g/kg in lambs, and 12 g/kg in adult guinea pigs.⁹ Colchicine is resistant to drying and heating. Although other alkaloids have

been isolated from *C. autumnale*, guinea pigs and mice fed colchicine and *C. autumnale* bulbs developed the same gross and histologic lesions,¹⁴ indicating colchicine is the primary toxicant. Colchicine acts by binding to tubulin, thus interrupting microtubule-dependent cell function. The actively dividing cells in the crypts of the intestine are the most sensitive cells to the effects of colchicine,^{3,14} although other microtubulin functions (such as inhibition of vesicle transport in axons) may be affected over time.³

Clinical signs change over time. Initially, diarrhea, often hemorrhagic, is seen as cell division in enterocytes is halted. Severe abdominal pain, vomiting, depression, and hypersalivation may also be present. These initial signs may not develop for 12 to 24 hours after ingestion because of the slow absorption of colchicine. Next (24 to 72 hours after ingestion), signs of weakness, ataxia, paresis, or collapse may be seen. In people, multiple organ failure (especially kidney and liver) is described.^{3,9} Alanine transaminase activity was elevated in a dog about 27 hours after it ingested *Gloriosa* species (ASPCA APCC Database: Unpublished data, 2002). Although little clinical information is available, clinical signs may last up to a week. In most cases, decontamination (emesis, activated charcoal) and symptomatic and supportive care effectively treat intoxication. But in severe cases, death may occur up to several days after ingestion.³

Cardiotoxic signs

Ingestion of *Convallaria majalis* (lily of the valley; Figure 2) has been associated with digitalis-like symptoms in people¹⁵ as well as sudden collapse and death in a dog.¹⁶ Diagnosis in the dog was based on the presence of leaf fragments in the small intestine, in addition to gross and histologic findings consistent with cardiac shock. *Convallaria* species contain many (about 40) cardenolides (cardiac glycosides containing a five-membered lactone ring); these are synthesized in leaves and stored in the rhizome.^{3,17} All portions of the plant are considered toxic, although the highest concentration of the cardenolides is in the rhizome.

Clinical signs are similar to those of digitalis toxico-

Continued

sis. Initially, GI signs are evident (vomiting and abdominal pain progressing to diarrhea, which may be hemorrhagic). Cardiovascular signs include bradycardia and other arrhythmias (including ventricular premature systole, paroxysmal tachycardia followed by complete heart block, and asystole). Affected animals may appear weak and depressed. Seizures, coma, and death may occur.^{3,18}

Initial treatment consists of detoxification. Induce emesis in asymptomatic animals, if not contraindicated. Administer activated charcoal (with a cathartic). Because of potential enterohepatic recirculation, repeated doses of activated charcoal may be appropriate. Administer GI protectants (e.g. sucralfate, H₂ antagonists) and antiemetics (metoclopramide) as appropriate to alleviate signs. Monitor symptomatic animals for cardiac arrhythmias and electrolyte and fluid imbalances. Closely monitor serum potassium concentrations, and treat hyperkalemia as needed. If you detect tachyarrhythmias, administer lidocaine or phenytoin. Propranolol would be the third choice of anti-arrhythmic, because it can cause severe bradycardia.⁶ Atropine to treat bradycardia or second- or third-degree atrioventricular block has also been recommended.^{3,6,18} Avoid calcium-containing fluids.¹²

For animals with severe cardiac signs, especially if the serum potassium concentration is elevated, digoxin-specific antigen binding fragments (Fab) derived from antidigoxin antibodies from sheep (Digibind—GlaxoSmithKline) may be considered. Although digoxin immune Fab (ovine) is formulated to bind digoxin (one vial contains 38 mg digoxin-specific Fab fragments—enough to bind about 0.5 mg digoxin), it is also effective for binding to other cardiac glycosides, such as those in *Convallaria* species. The dose of digoxin immune Fab is based on serum toxin concentrations. Since it's not possible to calculate the exact dose of digoxin immune Fab, begin with a small amount, and administer it to effect. The dose may need to be repeated if clinical signs return.¹⁹ Digoxin immune Fab is expensive, and several vials may be required for successful treatment.

Conclusion

The toxic effects of exposure to bulbous flowering plants vary from mild to potentially lethal. Effective treatment of an intoxication relies on knowing the plant species involved. With most of these plants, the prognosis is good with symptomatic treatment.

REFERENCES

1. Julian, C.G.; Bowers, P.W.: The nature and distribution of daffodil pickers' rash. *Contact Dermatitis* 37 (6):259-262; 1997.
2. Lamminpää, A. *et al.*: Occupational allergic contact dermatitis caused by decorative plants. *Contact Dermatitis* 34 (5):330-335; 1996.
3. Burrows, G.E.; Tyrl, R.J.: *Toxic Plants of North America*. Iowa State University Press, Ames, 2001; pp 105-111, 720-724, 751-805, 991-992.
4. Willemse, T.; Vroom, M.A.: Allergic dermatitis in a great Dane due to contact with hippeastrum. *Vet. Rec.* 122 (20):490-491; 1988.
5. Beasley, V.R.; Dorman, D.C.: Management of toxicoses. *Vet. Clin. North Am. (Small Anim. Pract.)* 20 (2):307-337; 1990.
6. Plumb, D.C.: *Veterinary Drug Handbook*, 3rd Ed. Iowa State University Press, Ames, 1999; pp 229-233, 418-420, 487-490.
7. Hill, L.; Hill, N.: Late bloomers. *Bulbs: Four Seasons of Beautiful Blooms*. Storey Books, Pownal, Vt., 1994; pp 159-160.
8. Bruce, E.A.; Agassiz, B.C.: Iris poisoning of calves. *JAVMA* 9:72-74; 1920.
9. Cooper, M.R.; Johnson, A.W.: *Poisonous Plants and Fungi in Britain*, 2nd Ed. The Stationery Office, London, England, 1998; pp 34-35, 153-155.
10. Litovitz, T.L.; Fahey, B.A.: Please don't eat the daffodils. *N. Engl. J. Med.* 306 (9):547; 1982.
11. Knight, M.W.; Dorman, D.C.: Selected poisonous plant concerns in small animals. *Vet. Med.* 92 (3):260-272; 1997.
12. Murphy, M.J.: Toxin exposures in dogs and cats: Pesticides and biotoxins. *JAVMA* 205 (3):414-421; 1994.
13. Lawrence, R.A.; Schneider, M.F.: The toxicity of household plants: A twenty-five-year prospective study (abst.). *Vet. Hum. Toxicol.* 29:467; 1987.
14. Yamada, M. *et al.*: Comparison of enterotoxicity between autumn crocus (*Colchicum autumnale* L.) and colchicines in the guinea pig and mouse: Enterotoxicity in the guinea pig differs from that in the mouse. *J. Vet. Med. Sci.* 62 (8):809-813; 2000.
15. Edgerton, P.H.: Symptoms of digitalis-like toxicity in a family after accidental ingestion of lily of the valley plant. *J. Emerg. Nurs.* 15 (3):220-223; 1989.
16. Moxley, R.A. *et al.*: Apparent toxicosis associated with lily-of-the-valley (*Convallaria majalis*) ingestion in a dog. *JAVMA* 195 (4):485-487; 1989.
17. Schrutka-Rechtenstamm, R. *et al.*: Bioconversions leading to minor cardiac glycosides in *Convallaria majalis*. *Phytochemistry* 25:1107-1109; 1986.
18. Osweiler, G.D.: Plant related toxicosis. *Toxicology*. Williams & Wilkins, Philadelphia, Pa., 1996; p 392.
19. GlaxoSmithKline: Digibind product information. Research Triangle Park, N.C., 2001. ■