Acute renal failure caused by lily ingestion in six cats

Cathy E. Langston, DVM, DACVIM

A 4-year-old castrated male domestic longhair cat (cat 1) was seen eating part of an Easter lily. The cat began vomiting 24 to 48 hours later, and the owner brought the cat to a local veterinarian for examination 3 to 4 days after it ingested the plant. At that time, the cat had a fever (rectal temperature, 40.2°C [104.4°F]), and acute renal failure (ARF) was diagnosed on the basis of high BUN (165 mg/dl; reference range, 15 to 34 mg/dl) and serum creatinine (10 mg/dl; reference range, 0.8 to 2.3 mg/dl) concentrations. The cat was hospitalized for treatment by the initial veterinarian, but BUN (165 mg/dl) and serum creatinine (9.0 mg/dl) concentrations were still high after 48 hours of IV fluid diuresis. At this time, the cat was vomiting and anorectic but still producing urine. The cat was referred to a second veterinary hospital for 24-hour care and, on initial examination at this hospital, was determined to be overhydrated and to have pulmonary edema. Both kidneys were larger than normal, and signs of pain were evident during palpation of the kidneys. Furosemide (2 mg/kg [0.9 mg/lb] of body weight, IV, q 6 h) was administered, and fluid therapy was discontinued. Twenty-four hours later, the azotemia had worsened (BUN, 202 mg/dl; creatinine, 14.6 mg/dl). Rare ventricular premature contractions were detected, and urine output had decreased from 1 ml/kg/h (0.45 ml/lb/h) to 0.5 ml/kg/h (0.23 ml/lb/h; reference range, 1 to 2 ml/kg/h [0.45 to 0.9 ml/lb/h]). A dopamine infusion (2 µg/kg/min [0.9 µg/lb/min]) was initiated, and the cat was referred to the Animal Medical Center for hemodialysis.

During initial examination at the Animal Medical Center, thoracic radiography revealed pulmonary edema and pleural effusion. One hundred thirty milliliters of transudate was removed via thoracocentesis. Intravenous administration of fluids and dopamine was discontinued, and hemodialysis was initiated. During the first hemodialysis treatment, a net volume of 388 ml of fluid was removed; an additional 84 ml of pleural effusion was removed by thoracocentesis. The following day (9 days after ingestion of the plant), a nasooesophageal feeding tube was placed, and the cat was fed a combination of 2 liquid diets formulated for cats with renal failure. The cat received 12 additional dialysis treatments during the next 30 days. Blood transfusions were performed 10 and 11 days after ingestion of the plant, and 12 days after ingestion of the plant, the cat was bright and responsive without any signs of abdominal or renal pain. The cat continued to make an unquantified amount of urine, and the azotemia was resolving as expected with hemodialysis.

Seventeen days after ingestion of the plant, the cat developed signs of abdominal pain, ascites, and chylous pleural effusion. Echocardiography revealed dilatation of the cardiac chambers with normal fractional shortening, consistent with volume overload. No thrombus was detected, although chylothorax has been seen in association with right atrial thrombosis in cats undergoing hemodialysis. Thoracocentesis was repeated as needed during the ensuing week. Triglyceride content of the fluid was 1,751 mg/dl (reference range, 20 to 90 mg/dl), and the cytologic appearance of the fluid was consistent with chylous effusion. Because of the chylous nature of the fluid, rutin, a benzopyrone thought to act by stimulating macrophage phagocytosis and protectolysis, was given at a dosage of 250 mg, PO, every 12 hours, starting on day 24.

Results of abdominal ultrasonography on days 22 and 29 were suggestive of pancreatitis. During hemodialysis on days 23 and 28, the cat developed hypotension that responded to treatment with small boluses (10 ml each) of saline solution and a temporary decrease in the rate of ultrafiltration. On day 29, the cat collapsed but responded to administration of fluids and oxygen; blood pressure was not recorded. Treatment with dopamine (5 µg/kg/min [2.3 µg/lb/min]) to control the hypotension was initiated, but on day 31, the cat developed cardiopulmonary arrest. Resuscitation efforts were discontinued at the owner’s request.

Necropsy revealed renal tubular necrosis with a moderate number of polarized crystals in the collecting tubules. There was chronic fibrosing pancreatitis with peripancreatic fat necrosis and steatitis. In addition, bacterial pleuritis, uremic pneumonitis, and a thrombus in the caudal vena cava were identified.

A 5-year-old spayed female domestic shorthair cat (cat 2) was brought to the referring veterinarian because of vomiting and lethargy, having eaten part of a tiger lily 2 days previously. The cat had severe azotemia (BUN, 240 mg/dl; creatinine, 39.1 mg/dl), glucosuria, and hematuria, and a diagnosis of ARF was made. The cat was hospitalized and responded to IV administration of fluids, and the cat was discharged with instructions that fluid treatment be continued (200 ml, SC, q 24 h). Blood urea nitrogen concentration at the time of discharge was 89 mg/dl; serum creatinine concentration was 10.5 mg/dl. Thirty-seven
days after ingestion of the plant, the cat was reexamined; BUN concentration was 64 mg/dl, and serum creatinine concentration was 8.6 mg/dl. Glucosuria persisted despite normoglycemia.

Forty-seven days after ingestion of the plant, the cat was examined by the emergency service at the Animal Medical Center because of vomiting. At that time, the cat was dehydrated, hypokalemic (2.87 mEq/L; reference range, 3.5 to 5.3 mEq/L), and anemic (PCV, 23%; reference range, 24 to 45%). The BUN concentration was 66 mg/dl, and the serum creatinine concentration was 6.1 mg/dl. Subjectively, it appeared that the cat had polyuria.

Abdominal ultrasonography revealed thick renal cortices with normal echogenicity. After rehydration, the PCV was 13%, and the cat received a blood transfusion. The cat was discharged after 4 days, with a BUN concentration of 79 mg/dl and a serum creatinine concentration of 4.2 mg/dl. Sixteen months after ingestion of the plant, the cat had a BUN concentration of 55 mg/dl and a serum creatinine concentration of 4.1 mg/dl. The BUN concentration was 11.4 mg/dl.

A 1-year-old spayed female domestic shorthair cat (cat 3) was examined by the referring veterinarian because of vomiting and lethargy. A diagnosis of ARF was made on the basis of azotemia (BUN, 223 mg/dl; creatinine, 24.2 mg/dl) and a history of Easter lily ingestion approximately 5 days previously, and the cat was referred to the Animal Medical Center for treatment.

At the time of referral to the Animal Medical Center, the cat was dehydrated and polyuric (urine output, 7 ml/kg/h), with renomegaly and signs of renal pain. The cat had mild metabolic acidosis (venous pH, 7.235; reference range, 7.35 to 7.45) and mild hypokalemia (3.42 mmol/L) and was mildly hypotensive (Doppler systolic blood pressure, 70 mm Hg) but was still eating and drinking. Intravenous fluid therapy was instituted to correct dehydration and hypotension, provide for maintenance needs, and promote diuresis. Uremia was managed with ranitidine (2.2 mg/kg [1 mg/lb], IV, q 24 h) and aluminum hydroxide (64 mg, PO, q 8 h). A fever (rectal temperature, 39.8 C [103.6 F]) that developed on day 9 was treated with ampicillin (22 mg/kg [10 mg/lb], IV, q 8 h) and resolved by day 13. Azotemia gradually decreased, and the cat was discharged 21 days after ingestion of the plant; BUN concentration was 84 mg/dl, and serum creatinine concentration was 6.5 mg/dl. Medications prescribed at the time of discharge were lactated Ringer’s solution (125 ml, SC, q 24 h), aluminum hydroxide (128 mg, PO, q 8 h), famotidine (2.5 mg, PO, q 24 h), a multivitamin with iron (2 ml, PO, q 12 h), amoxicillin (100 mg, PO, q 12 h), and cyproheptadine (2 mg, PO, q 12 h). During a recheck examination 4 days after discharge, the owner reported that the cat was doing well and had gained 0.1 kg (0.2 lb). The BUN concentration was 93 mg/dl, and the serum creatinine concentration was 5.5 mg/dl. According to the owner 19 months later, the cat was doing well and was not receiving any treatment other than being fed a restricted protein diet.

A 1.5-year-old castrated male domestic shorthair cat (cat 5) was examined by the referring veterinarian because of lethargy; the owner reported that the cat had vomited Easter lily leaves 4 days previously. At the time of examination by the referring veterinarian, the cat was found to have oliguria, renomegaly, and signs of abdominal pain. The cat had vomited Easter lily at the same time that cat 3 did. A diagnosis of ARF with severe azotemia (BUN, 265 mg/dl; creatinine, 24.3 mg/dl) was made, and the cat was referred for treatment.

On initial examination at the Animal Medical Center, the cat was dehydrated and had renomegaly, signs of renal pain, polyuria (urine output, 5 ml/kg/h [2.3 ml/lb/h]), severe metabolic acidosis (venous pH, 7.080), and hypocalcemia (ionized calcium, 2.4 mg/dl; reference range, 4 to 6 mg/dl). The cat was treated with fluids IV; ranitidine (2.2 mg/kg [1 mg/lb], IV, q 24 h) and aluminum hydroxide (6+ mg, PO, q 8 h) were administered because of the uremia. Azotemia gradually decreased with treatment, but not as dramatically as in cat 3. A blood transfusion was given 14 days after ingestion of the plant because of anemia (PCV, 13%) and clinical signs attributable to anemia (heart murmur and tachycardia). Treatment with ampicillin (22 mg/kg [10 mg/lb], IV, q 8 h) was instituted on day 14, because results of bacterial culture of a urine sample obtained at the time an indwelling urinary catheter was removed were positive. The cat was force-fed during much of the hospitalization period and lost 2 pounds during this time. The cat began eating on its own on day 17 after treatment with cyproheptadine (2 mg, PO, q 12 h). During a recheck examination 4 days after discharge, the owner reported that the cat was doing well and had gained 0.1 kg (0.2 lb). The BUN concentration was 93 mg/dl, and the serum creatinine concentration was 5.5 mg/dl. According to the owner 19 months later, the cat was doing well and was not receiving any treatment other than being fed a restricted protein diet.

A 1-year-old castrated male domestic shorthair cat (cat 4), a littermate of cat 3 owned by the same individual, was examined by the referring veterinarian because of vomiting and lethargy approximately 5 days after Easter lily ingestion. Cat 4 had eaten the Easter lily at the same time that cat 3 did. A diagnosis of ARF with severe azotemia (BUN, 265 mg/dl; creatinine, 24.3 mg/dl) was made, and the cat was referred for treatment.

On initial examination at the Animal Medical Center, the cat was dehydrated and had renomegaly, signs of renal pain, polyuria (urine output, 5 ml/kg/h [2.3 ml/lb/h]), severe metabolic acidosis (venous pH, 7.080), and hypocalcemia (ionized calcium, 2.4 mg/dl; reference range, 4 to 6 mg/dl). The cat was treated with fluids IV; ranitidine (2.2 mg/kg [1 mg/lb], IV, q 24 h) and aluminum hydroxide (6+ mg, PO, q 8 h) were administered because of the uremia. Azotemia gradually decreased with treatment, but not as dramatically as in cat 3. A blood transfusion was given 14 days after ingestion of the plant because of anemia (PCV, 13%) and clinical signs attributable to anemia (heart murmur and tachycardia). Treatment with ampicillin (22 mg/kg [10 mg/lb], IV, q 8 h) was instituted on day 14, because results of bacterial culture of a urine sample obtained at the time an indwelling urinary catheter was removed were positive. The cat was force-fed during much of the hospitalization period and lost 2 pounds during this time. The cat began eating on its own on day 17 after treatment with cyproheptadine (2 mg, PO, q 12 h) was initiated. The cat was discharged 21 days after ingestion of the plant; BUN concentration was 84 mg/dl, and serum creatinine concentration was 6.5 mg/dl. Medications prescribed at the time of discharge were lactated Ringer’s solution (125 ml, SC, q 24 h), aluminum hydroxide (128 mg, PO, q 8 h), famotidine (2.5 mg, PO, q 24 h), a multivitamin with iron (2 ml, PO, q 12 h), amoxicillin (100 mg, PO, q 12 h), and cyproheptadine (2 mg, PO, q 12 h). During a recheck examination 4 days after discharge, the owner reported that the cat was doing well and had gained 0.1 kg (0.2 lb). The BUN concentration was 93 mg/dl, and the serum creatinine concentration was 5.5 mg/dl. According to the owner 19 months later, the cat was doing well and was not receiving any treatment other than being fed a restricted protein diet.
Animal Medical Center the following day for additional treatment.

On initial examination, the cat was overhydrated and anuric and had pleural effusion. The anuria was unresponsive to treatment with furosemide (2 mg/kg [0.9 mg/lb], IV, initially; then 4 mg/kg [1.8 mg/lb], IV, 2 hours later). The hyperkalemia was treated with sodium bicarbonate (6 mEq, IV, administered as a bolus over 15 min). Hemodialysis was performed the day of admission to the Animal Medical Center and was repeated on days 6, 7, and 8. Immediately after the hemodialysis treatment on day 6, the cat became agitated and was treated with mannitol, IV, for presumptive dialysis disequilibrium syndrome (DDS), a condition caused by rapid changes in serum osmolality leading to cerebral edema. The agitation lessened within 30 minutes. On day 10, the cat had a seizure. Systolic blood pressure was 216 mm Hg, and iridial hemorrhage was evident. Treatment with amlodipine was started, and hemodialysis was performed a fifth time. On day 11, signs of abdominal pain had resolved, but the cat had a fever (rectal temperature, 39.6°C) that resolved without treatment. On day 12, a nasojejunal feeding tube was placed, and the cat was fed a liquid diet. On day 13, the fever recurred, and there was difficulty obtaining adequate blood flow from the dialysis catheter during hemodialysis. On day 14, the fever persisted, but the cat began producing urine after 9 days of anuria. On day 15, a hypotensive episode occurred, followed by vomiting and respiratory arrest. The cat was resuscitated, and hemodialysis was performed, but the cat had neurologic deficits consistent with a left cerebral lesion attributable to a cerebrovascular accident. On day 16, the cat suffered cardiopulmonary arrest that was not responsive to resuscitation efforts.

Necropsy revealed renal tubular necrosis with multifocal mineralization of the tubules. Regenerative epithelial cells were evident, and no crystals were seen with polarized light. There was severe acute-to-subacute fibrosing pancreatitis with marked fat saponification and ascites, along with pulmonary edema and increased numbers of alveolar macrophages. A thrombus containing a mixed population of bacteria was associated with the distal tip of the dialysis catheter. At the owner’s request, the CNS was not examined.

A 5-year-old castrated male domestic shorthair cat (cat 6) was examined at the Animal Medical Center 2 days after ingesting a tiger lily leaf and stem. The cat had been anorectic and lethargic since ingesting the plant parts, with no evidence of urinary production. On initial examination, the cat appeared painful during abdominal palpation and had a BUN concentration of 158 mg/dL, a serum creatinine concentration of 11.2 mg/dL, and a serum potassium concentration of 8.1 mEq/L. The cat was treated with saline (0.9% NaCl) solution, IV (50 ml for the first hour, then 5 ml/h), regular insulin to treat the hyperkalemia (1 unit, 2 doses), dextrose as a substrate for the insulin (1.5 g, 2 doses), and furosemide (13.3 mg boluses, q 1 h) but did not respond to treatment and was euthanatized at the owner’s request 9 hours after treatment was started. The owner declined necropsy.

Acute renal failure can be caused by a variety of toxic substances. The most notable of these is ethylene glycol, but a large variety of plants can also cause renal damage. Oxalate-containing plants, such as dumbcane (Difenbahia spp), philodendron (Philodendron spp), peace lily (Spathiphyllum spp), and devil’s ivy (Epipremnum aureum), cause stomatitis, which typically limits the amount of plant material ingested, but renal failure can develop if a sufficient quantity is ingested. This is a problem primarily with philodendron ingestion. Other oxalate-containing plants of toxicologic interest primarily for livestock include beets (Beta vulgaris), lamb’s quarters (Chenopodium album), Halogeton glomeratus, rhubarb (Rheum rhaponticum), and greaseweed (Sarcobatus vermiculatus). Renal failure can also result from cholecalciferol toxicity associated with ingestion of night-blooming jasmine (Cestrum diurnum) or Solanum malacoxylon (a member of the Solanaceae or nightshade family). Redroot pigweed (Amaranthus retroflexus) causes renal failure with perirenal retroperitoneal edema, but toxic effects are primarily seen in swine and cattle. Oak poisoning can cause renal failure in cattle and horses.

Members of the lily family (Liliaceae) can cause a variety of toxic signs when ingested by pets or livestock. Lily-of-the-valley (Convallaria majalis) contains colchicines, which lead to liver disease, but toxics are seen primarily in livestock. Bluebell bulb (Hyacinthoides nonscripta) poisoning in horses can cause anuria. Garlic and onion (Allium spp) can cause hemolytic anemia in dogs and cats. Easter lily (Lilium longiflorum), day lily (Hemerocallis spp), tiger lily (Lilium spp), Japanese show lily (L. hybridum), and rubrum lily (L. rubrum) can all cause renal toxicosis in cats.

The present report describes findings for 6 cats that developed ARF after known or suspected ingestion of lily plants. Renal failure in all of these cats was severe, with a mean BUN concentration of 215 mg/dL (median, 232 mg/dL) and a mean serum creatinine concentration of 22.3 mg/dL (median, 24.3 mg/dL) 2 to 5 days after ingestion of the plants. High serum creatinine concentrations in cats with lily toxicosis have been reported previously. Vomiting and lethargy were the most common initial clinical signs reported by owners of cats in the present report and developed between 1 and 3 days after exposure. In most cats, renal failure was evident at the time signs were first noticed. Gastrointestinal tract upset, lethargy, and anorexia reportedly can also develop during the early stages after ingestion, although vomiting in the cats in the present report could have been secondary to uremia.

Of the 6 cats in the present report, 2 had anuria, 1 had oliguria, and 2 had polyuria. Initial urine output was not determined in the remaining cat, although the cat was polyuric 1 month after ingestion. The reason for this variation in urine output is unclear. For some nephrotoxins, the severity of renal failure is dose dependent, with polyuric renal failure representing less severe toxicosis. The exact amount of plant materi-
al ingested by each cat was unknown. In addition, potency may vary at different growth stages or in different parts of the plant, although both leaves and flowers are known to be toxic. Host factors may also be involved, including amount of gastrointestinal tract absorption or renal susceptibility.

Results for these cats suggest that recovery from lily toxicosis is possible if adequate supportive care can be given. In 2 of the 3 cats with oliguria or anuria, urine production improved with treatment, and this increase in urine production likely represented some recovery of renal function. In cat 5, there were regenerative epithelial cells in the renal tubules 16 days after plant ingestion.

Cat 5 had substantial neurologic abnormalities; however, these abnormalities did not develop until after dialysis was started and could be attributed to DDS, hypertensive encephalopathy, uremic encephalopathy, or a cerebrovascular accident. Nevertheless, it is uncertain whether the neurologic problems were related exclusively to the uremia and its treatment or whether there was some toxic damage to the CNS that increased susceptibility to other insults. However, Easter lily toxicosis has not been previously associated with neurologic disorders.

Pancreatitis was detected in both cats in which a necropsy was performed, suggesting that pancreatitis may be a complication of lily exposure. Signs consistent with pancreatitis were first noticed 13 and 17 days after plant ingestion, and in cat 1, pancreatitis was the likely cause of death. In cat 5, it could not be determined whether pancreatitis, septicemia, neurologic abnormalities, or pulmonary disease was the probable cause of death. Both cats, however, had undergone hemodialysis, and in the author’s experience, pancreatitis has been identified infrequently in other cats undergoing dialysis, particularly cats with ethylene glycol-induced ARF. Whether pancreatitis is attributable to a toxic plant component, secondary to a uremic toxin, induced by some factor involved in the dialysis process, or a result of some other factor is unclear. In the 3 cats that survived, no clinical signs of pancreatitis were reported, although no diagnostic evaluations specific for pancreatitis were attempted.

Dialysis was essential in the management of 2 cats in the present report. Both were unresponsive to medical treatment for ARF and had life-threatening signs of uremia (ie, hyperkalemia and volume overload) that resolved with dialysis. Even though the outcome of these 2 cats was poor, it is impossible to directly compare results of medical management with results of hemodialysis, as cats that underwent hemodialysis were more severely affected. Dialysis is the only treatment that can rapidly resolve life-threatening signs of uremia and allow the cat time to regenerate renal tubules. Dialysis has been shown to provide renal support in other nephrotoxic diseases, such as ethylene glycol poisoning in cats, with 44% of cats that undergo dialysis surviving this otherwise lethal form of renal failure.

Peritoneal dialysis should be considered if hemodialysis is not available.

The outcome of lily toxicoses in these cats was not favorable. Three cats died or were euthanized, and the remaining 3 developed chronic renal failure.

However, results for these cats do suggest that ARF secondary to lily ingestion is not uniformly fatal, as has previously been reported. None of the cats in the present report were examined early enough for decontamination of the gastrointestinal tract (ie, induction of emesis and gastric lavage with activated charcoal) to be helpful. However, it has been reported that if decontamination is performed within 6 hours of plant ingestion, renal failure will not ensue.

In addition to Easter lily and tiger lily, many types of lily plants can cause renal failure. Toxicosis is not limited to the time around Easter and should be considered in cats with clinical signs suggestive of ARF. Common findings for cats in the present report included vomiting and lethargy 1 to 5 days after plant ingestion, severe azotemia, renomegaly, and signs of abdominal pain. These findings should prompt clinicians to inquire about the potential for exposure to lilies. Although the toxic principle of lilies that is associated with renal damage is unknown, the amount of damage to the kidneys from lily toxicosis seems substantial. The type of treatment necessary depends on the severity of signs, but with intensive treatment, partial recovery of renal function is possible in some cases.

References