Outcome following gastrointestinal tract decontamination and intravenous fluid diuresis in cats with known lily ingestion: 25 cases (2001–2010)

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Objective—To describe the outcome of cats treated with gastrointestinal tract decontamination, IV fluid diuresis, or both after ingestion of plant material from lilies of the *Lilium* and *Hemerocallis* genera.

Design—Retrospective case series.

Animals—25 cats evaluated after ingestion of lily plants.

Procedures—Medical records of cats examined at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania with known lily ingestion between July 2001 and April 2010 were reviewed. Inclusion in the study required evidence of lily plant ingestion within the preceding 48 hours. Type of lily ingested, time of ingestion, gastrointestinal tract decontamination procedures performed, and IV fluid diuresis were recorded. The presence or absence of acute kidney injury was determined by evaluating BUN concentration, creatinine concentration, and urine specific gravity. Outcome was defined as survival to discharge, death, or euthanasia.

Results—The time from ingestion until evaluation at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania ranged from < 30 minutes to 48 hours. Nineteen cats received gastrointestinal tract decontamination (18 cats at our hospital and 1 cat by the referring veterinarian). Twenty-three cats were admitted to the hospital for IV fluid diuresis, supportive care, and monitoring. Seventeen of these 23 (74%) cats had normal BUN and creatinine concentrations throughout hospitalization. At the time of discharge from the hospital, 2 of the 23 (9%) hospitalized cats had an increased BUN concentration, creatinine concentration, or both. All 25 (100%) cats survived to discharge from the hospital.

Conclusions and Clinical Relevance—In this series of cats treated with gastrointestinal tract decontamination, IV fluid diuresis, or both within 48 hours after lily ingestion, the outcome was good, with a low incidence of acute kidney injury. Future studies are needed to determine the most effective gastrointestinal tract decontamination procedures and optimal duration of IV fluid therapy. (*J Am Vet Med Assoc* 2013;242:1110–1116)

Ingestion of plant material from lilies of the *Lilium* and *Hemerocallis* genera is known to cause AKI in cats, with the devastating, potentially fatal effects well documented in the veterinary literature.[1–4,a] The clinical signs of lily toxicosis typically include gastrointestinal tract signs (eg, vomiting, ptyalism, anorexia, and diarrhea), signs attributable to AKI (ie, polydipsia, polyuria, oliguria, anuria, renomegaly, signs of abdominal or renal pain, and oral ulceration), and nonspecific signs such as dehydration, hypothermia or fever, lethargy, depression, ventricular premature contractions, and death.[1–10,a] Neurologic signs (eg, ataxia, disorientation, head pressing, tremors, and seizures),[1,3,6,11] pancreatic degeneration or pancreatitis,[3,11] and paw and facial edema[6] have also been reported. Expected clinical pathological testing results from affected cats reflect AKI and include azotemia, with some authors reporting a disproportionately elevated creatinine concentration,[5,7–9] hyperphosphatemia, hypokalemia or hyperkalemia, hypocalcemia or hypercalcemia, anemia, metabolic acidosis, glucosuria, proteinuria, hematuria, decreased USG, and the presence of epithelial and granular casts in the urine.[2–4,6–8,a] Others have reported various elevations in hepatobiliary biomarkers, including activities of alanine transaminase, aspartate transaminase, alkaline phosphatase, and total bilirubin concentration.[2,5,6]

Abbreviations

AKI	Acute kidney injury
NAPCC	National Animal Poison Control Center
TS	Total solids
USG	Urine specific gravity

To our knowledge, the toxic component has not been identified, and it is unknown whether it is the same in both the *Lilium* and *Hemerocallis* genera. In a single experimental study[11] of Easter lily (*Lilium longiflorum*) toxicosis, cats were administered extracts of lily plant material via nasogastric tube to not only identify the toxic component in lilies but also determine diagnostic markers and assess pathological effects. It was

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determined that the toxic component is water soluble, is present in both the leaves and the flowers, and is more potent in the flowers. However, the exact toxin was not identified. Manifestations of lily toxicosis in this experimental study[11] included nephrotoxicosis, pancreatic degeneration, seizures, and death within hours after toxin administration (cause unknown).

Currently, the recommended treatment for lily ingestion in cats includes minimizing toxin absorption and supporting renal function through gastrointestinal tract decontamination procedures and IV fluid diuresis.[1,4,5,7–9] However, these recommendations appear to be speculative, with evidence for these treatments or any other treatment protocol lacking. To our knowledge, the only published data evaluating the efficacy of decontamination treatment for lily toxicosis are 2 small studies.[1,a] One study,[a] reported only as an abstract, evaluated the outcome of 13 cats with lily ingestion. Four cats decontaminated via emesis, activated charcoal, saline (0.9% NaCl) solution cathartic, and diuresed with fluids within 6 hours of Easter lily ingestion did not develop renal failure. Conversely, 4 cats in which intoxication was recognized > 18 hours after exposure all developed renal failure and had a 100% mortality rate. This abstract also reported an additional 5 previous cases in which the mortality rate was 100%; however, decontamination procedures or additional supportive treatment of these cats was not discussed. The other study,[1] which retrospectively evaluated 22 cats with daylily (*Hemerocallis*) toxicosis, briefly discusses decontamination and treatment measures for 4 cats with only gastrointestinal signs following lily ingestion. In these cases, activated charcoal was administered and IV fluid diuresis and supportive care were initiated within 24 to 36 hours after exposure. None of these 4 cats developed any detectable renal damage, and all made a full recovery.

Anecdotally, it has been our clinical experience that cats receiving gastrointestinal tract decontamination and IV fluid diuresis following lily ingestion rarely develop evidence of AKI and have a good outcome. Therefore, the purpose of the study reported here was to describe the outcome of cats that received gastrointestinal tract decontamination and IV fluid diuresis after ingestion of plant material from lilies of the *Lilium* and *Hemerocallis* genera.

Materials and Methods

Medical records at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania were searched for cats examined because of lily ingestion between July 2001 and April 2010. Each patient's medical record was examined to determine eligibility for inclusion. Cats were eligible for inclusion in the study if the cat was seen chewing on or ingesting any part of a lily plant, if there was evidence of a chewed lily plant (and only 1 animal had access to the plant), or if there was lily plant material found within the patient's oral cavity or vomitus. Patients were excluded if there was not definitive evidence of lily ingestion or ingestion had occurred > 48 hours prior to initial examination at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania.

The following data were recorded: breed, sex, age, admission date, time since lily ingestion, gastrointestinal tract decontamination procedures performed both prior to and at admission to the hospital, initial clinical pathology testing results (PCV and TS, BUN, and creatinine concentration), subsequent renal values (BUN and creatinine concentrations), urinalysis results, medications administered, IV fluid therapy (type and length), duration of hospitalization, outcome, and follow-up information (if available). Outcome was defined as survival to discharge, death, or euthanasia. The data collected were analyzed for cats with evidence of AKI, which, for the purposes of this study, was defined as the presence of concurrent azotemia and USG < 1.035.

To evaluate whether the length of time that had elapsed since lily ingestion and initiation of decontamination procedures affected outcome, the cats were divided into 2 groups. Groups were defined on the basis of the time between ingestion of the lily plant and admission to the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania. Group 1 was defined as patients with a history of ingestion within the preceding 6 hours (≤ 6 hours), and group 2 was defined as patients with a history of ingestion from > 6 hours to 48 hours prior.

Results

An initial search of medical records identified 28 cats with known lily ingestion. However, after the inclusion and exclusion criteria were applied, 3 cats were eliminated from further analysis because the lily ingestion occurred > 48 hours prior. Therefore, 25 cats met the study inclusion criteria. Twenty-one cats were domestic shorthair, 2 were domestic longhair, 1 was a Siamese, and 1 was a Ragdoll. Fifteen cats were spayed females, and 10 were castrated males. The median age was 2.5 years (range, 0.33 to 9.92 years).

The type of lily ingested was known for 17 cats: 6 cats ingested Oriental–Oriental hybrid lilies, 6 cats ingested Asiatic–Asiatic hybrid lilies (including 2 cats that had ingested tiger lilies), 3 cats ingested Easter lilies, and 2 cats ingested daylilies. The type of lily ingested was unknown in 8 cats. Oriental, Easter, and Asiatic lilies are all from the *Lilium* genus, whereas daylilies are from the *Hemerocallis* genus. The lilies had been ingested throughout most months of the year (excluding only January, September, and December), with the greatest number ingested in February, March, and May, with 4 cases seen in each of these months.

The time between ingestion of lily plant and initial evaluation at the Matthew J. Ryan Veterinary Hospital ranged from shortly after witnessed ingestion (< 30 minutes) to 48 hours. There were 17 cats in group 1 (ingestion of lily within ≤ 6 hours) and 8 cats in group 2 (ingestion of lily between > 6 and 48 hours). Within group 2, 2 cats were examined between > 6 and 18 hours after ingestion and 6 cats > 18 hours after ingestion, although it should be noted that the times of ingestion in group 2 were generally less exact than those in group 1.

Nineteen of 25 (76%) cats received gastrointestinal tract decontamination (18 cats treated at our hospital and 1 cat treated by the referring veterinarian), includ-

ing all 17 cats from group 1 and 2 of 8 cats from group 2. Gastrointestinal tract decontamination procedures included induction of emesis, gastric lavage, administration of activated charcoal, or a combination of these treatments. Six cats, all in group 2, did not receive any form of gastrointestinal tract decontamination. Eight cats (3 in group 1 and 5 in group 2) vomited at home prior to evaluation. Lily plant material was identified in the vomitus of 5 of these cats (3 in group 1 and 2 in group 2). Two of the cats that vomited hours prior to hospital admission were placed in group 2 on the basis of the established criteria (both had vomitus containing lily leaves or petals). However, both of these cats likely vomited within 6 hours after lily ingestion. Of these 8 cats, 5 went on to have further decontamination procedures: 2 cats had emesis induction, 2 cats had emesis induction and activated charcoal administration, and 1 cat was administered activated charcoal via orogastric tube by the primary care veterinarian prior to referral. No additional lily plant material was reported to be found in any of the cats in which emesis was induced again. Finally, 1 group 1 cat had a piece of lily leaf extracted from the back of the oropharynx during its physical examination.

Of the 19 cats receiving gastrointestinal tract decontamination, 5 had emesis induction alone; 8 had emesis induction followed by administration of activated charcoal; 1 had emesis induction followed by gastric lavage; 3 had emesis induction, gastric lavage, and activated charcoal administration; and 2 had only activated charcoal administration.

Of the 17 cats in which emesis was induced at our hospital, all were administered an α_2-adrenoceptor agonist. Sixteen were administered xylazine (0.4 to 0.5 mg/kg [0.18 to 0.23 mg/lb], IM [except in 1 case in which the route of administration was not recorded]), and 1 was administered dexmedetomidine (35.8 µg/kg [16.3 µg/lb], IM). Four of these cats were administered 2 emetics because of unsuccessful emesis induction with the initial drug: hydrogen peroxide was administered in 3 cats, and apomorphine was administered in 1. None of these cats responded to the second emetic, and gastric lavage was performed in all 4. There were an additional 2 cats that were administered hydrogen peroxide prior to initial evaluation at our hospital (unsuccessful in 1 cat and of unknown efficacy in the other). Both of these cats were administered xylazine at our hospital, which resulted in successful emesis induction.

Emesis induction was successful in 8 of 17 cats (including 1 cat that had vomited prior to evaluation), unsuccessful in 7 (including 2 cats that had vomited prior to evaluation), and of unknown efficacy in 2 (including 1 cat that had vomited prior to evaluation). Of the 8 cats in which emesis induction was successful, identifiable lily plant material was recovered in 3 cases. Of the 4 cats that had gastric lavage performed, only 1 had identifiable lily plant material recovered. The overall rate of lily plant material recovery was 10 of 25 cats. This includes the 5 cats that had self-decontaminated prior to hospital admission, the 1 cat in which lily plant material was removed from the oropharynx, and the 4 cats that had lily plant material recovered via emesis induction or gastric lavage.

Twenty-three cats were hospitalized because of lily ingestion: 16 of 17 from group 1 and 7 of 8 from group 2. All 23 cats survived to discharge. The length of hospitalization ranged from approximately 7.5 to 151.5 hours (median, 46 hours). All hospitalized cats received IV fluid diuresis with isotonic crystalloid fluids, with the rate of administration ranging from 0.9 to 10.2 mL/kg/h (0.4 to 4.6 mL/lb/h). However, the rate of IV fluid administration did vary over the course of hospitalization. The duration of administration of IV fluids was determined by the primary veterinarian managing the case and ranged from approximately 4.5 to 92 hours (median, 43.5 hours). In addition to isotonic crystalloid fluids, 1 cat received a whole blood transfusion and a synthetic colloid (6% hetastarch in saline solution) because of anemia that developed secondary to hemorrhage from the IV catheter during inadvertent fluid line disconnection. Three cats (1 from group 1 and 2 from group 2) were known to have received fluid therapy prior to referral to the Matthew J. Ryan Veterinary Hospital. Intravenous fluids were administered in 2 cases (1 from each of groups 1 and 2) and SC fluids in the third case (group 2).

The most commonly prescribed medication in the hospitalized cats was famotidine (16/23). Other medications administered included opioids (methadone [n = 1], butorphanol [1], buprenorphine [1], and fentanyl [1]), aluminum hydroxide (1), ranitidine (2), metoclopramide (1), ondansetron (1), antimicrobials (ampicillin and enrofloxacin followed by amoxicillin and clavulonic acid [1]), furosemide (2), azodyl (2), mirtazapine (1), enalapril (1), and amlodipine (1). Two cats also received medications for unrelated, preexisting conditions (atenolol and acetylsalicylic acid [n = 1] and pancreazyme [1]). Six hospitalized cats did not receive any medications.

Initial clinical pathology laboratory testing, including PCV and TS, BUN, and creatinine concentrations, was completed in all 25 cats (**Table 1**). At this time, 7 cats had

Table 1—Results of clinical laboratory testing in cats treated with gastrointestinal tract decontamination and IV fluid diuresis after ingestion of plant material from lilies of the *Lilium* and *Hemerocallis* genera between 2001 and 2010.

Variable	No. of cats	Results	No. above reference range	No. within reference range	No. below reference range	Reference range
Initial BUN concentration (mg/dL)	25	14 to > 100	5	19	1	15 to 32
Highest BUN concentration during hospitalization (mg/dL)	23*	18 to > 100 or 110	4	19	0	15 to 32
BUN concentration at discharge (mg/dL)	23*	10 to 110	1	10	12	15 to 32
Initial creatinine concentration (mg/dL)	22	0.9 to 3.1	1	19	2	1.0 to 2.0
Highest creatinine concentration measured (mg/dL)	21*	1.1 to 3.1	3	18	0	1.0 to 2.0
Creatinine concentration at discharge (mg/dL)	20*	0.8 to 2.5	2	16	2	1.0 to 2.0
PCV (%)	25	21 to 52	7	17	1	25 to 45

*Two cats were not admitted to the hospital and were not included.

an elevated PCV (46% to 52%; reference range, 25% to 45%) and 1 cat was anemic (PCV, 21%). Two cats had mildly decreased TS concentration (5.4 to 5.8 g/dL; reference range, 6.0 to 8.6 g/dL). Five (20%; 4 from group 1 and 1 from group 2) cats had an elevated BUN concentration (33 to > 100 mg/dL; reference range, 15 to 32 mg/dL). However, only 1 cat (group 1) had an elevated creatinine concentration (3.1 mg/dL; reference range, 1.0 to 2.0 mg/dL).

All 23 hospitalized cats had serial measurements of BUN and creatinine, with these values measured between 1 and 9 times (median, 3) throughout the course of hospitalization (Table 1). The median time the last laboratory testing was performed was 42.5 hours after admission (range, 7.5 to 143 hours). Seventeen (74%; 11 from group 1 and 6 from group 2) hospitalized cats never developed any increase in BUN or creatinine concentration. Six hospitalized cats (6/23 [26%]; 5 group 1 and 1 group 2) had a measured elevation of either BUN or creatinine concentration on at least 1 occasion. Of these cats, 3 (2 from group 1 and 1 from group 2) had a mildly elevated BUN concentration (33 to 36 mg/dL) at admission that subsequently normalized but no elevation in creatinine concentration either at admission or during the course of hospitalization. One cat (group 1) that had a normal creatinine concentration at admission had a single mildly elevated creatinine concentration (2.1 mg/dL) within the first 24 hours of hospitalization, but subsequently determined creatinine concentrations were normal (25 and 42.5 hours after admission). Another cat (group 1) developed an elevated creatinine concentration 11.5 hours after admission, which remained persistently elevated at 64.5 hours after admission (2.5 mg/dL at this time). After discharge, this cat had laboratory test monitoring performed through its primary care veterinarian for approximately 2.67 years. During this time, the BUN concentration ranged between 17 and 27 mg/dL, and the creatinine concentration between 1.4 and 2.7 mg/dL. Urine specific gravities were obtained approximately 7 months and 1.75 years after admission; USGs at both of these time points were adequately concentrated (1.045 and 1.052, respectively). This cat was treated with SC fluids and a renal diet for > 1 year and was still alive and clinically doing well approximately 6 years after initial evaluation. Finally, the 1 cat (group 1) that had elevated BUN and creatinine concentrations on initial evaluation continued to have elevated values (BUN concentration, 110 mg/dL; creatinine concentration, 2.1 mg/dL) at the time of the last laboratory testing (approx 143 hours after admission). This cat was managed for chronic renal failure for 106 days following initial evaluation, at which time it was euthanized owing to a combination of congestive heart failure and chronic renal failure.

Hospitalization was declined in 2 cases; these cats (1 from each of groups 1 and 2) were discharged without further treatment. The cat from group 1 had a mildly elevated BUN concentration (35 mg/dL) but normal creatinine concentration at this time. This cat was seen by the primary care veterinarian 2 days later and was found to have a normal BUN and creatinine concentration (24 and 1.3 mg/dL, respectively). Further laboratory testing was not performed; however, the cat was seen by the primary care veterinarian for dental prophylaxis approximately 3.25 years after the lily ingestion and continued to do well at last follow-up. The cat from group 2 had normal BUN and creatinine concentration and was hospitalized the following day at its primary care veterinarian's clinic for 12 to 24 hours; however, the details of treatments administered could not be obtained. Again, approximately 4 years after lily ingestion, the owner reported that the cat was doing well.

Of the 7 cats that had an elevated BUN or creatinine concentration recorded either at the initial consultation or throughout the hospitalization period, a USG was available for only 4. Three cats had a USG < 1.035 recorded; however, these samples were collected at various time points and may have been affected by fluid therapy or medications administered. In 1 cat, a USG of 1.006 was recorded after fluid therapy had been initiated. In the second cat, a USG of 1.030 was recorded; however, it could not be determined whether it was obtained before or after fluid therapy was initiated. In the third cat, a USG of 1.019 was obtained 1 hour after initial evaluation. This cat had an elevated BUN and creatinine concentration on initial evaluation and at the time of the last laboratory testing (143 hours). Finally, the fourth cat with a USG recorded had concentrated urine (USG > 1.050) prior to initiation of fluid therapy. This cat had a mildly elevated BUN concentration (36 mg/dL) on initial laboratory testing, but normal BUN and creatinine concentrations were measured during the remaining hospitalization period.

Discussion

In this series of cats with known lily ingestion treated at a large urban teaching hospital receiving gastrointestinal tract decontamination and IV fluid diuresis within 48 hours after lily ingestion, the outcome was good, with a low incidence of AKI. All 25 (100%) cats survived, and at the time of discharge from the hospital, only 2 of the 23 (9%) hospitalized cats had an increased BUN concentration, creatinine concentration, or both. Ingestion of Easter lily resulting in acute renal failure in cats was first noted by the NAPCC in 1989.[a] In the first published abstract[a] of 13 cats with Easter lily ingestion (1992), only 4 cats survived. Since that initial report, an additional 36 cats with known, nonexperimental lily ingestion (24 cases of *Hemerocallis* ingestion, 9 cases of *Lilium* ingestion, and 3 cases of lily ingestion of unknown genus) have been reported in the veterinary literature.[1–4,6,12]

Prior to the present study, information about the effect of early gastrointestinal tract decontamination and IV fluid diuresis in cats after lily ingestion was limited. In the initial abstract published by the NAPCC,[a] the only 4 surviving cats (of 13) were treated with emesis, activated charcoal, saline solution cathartic, and fluid diuresis within 6 hours after Easter lily ingestion. On the basis of this single abstract, gastric decontamination and IV fluid diuresis were initially recommended to be commenced within 6 hours after ingestion to prevent the development of AKI. However, the American Society for the Prevention of Cruelty to Animals Animal Poison Control Center has since expanded this opportune period of time to initiate treatment to 18 hours.[8]

However, to our knowledge, there is neither clinical nor experimental evidence published in the veterinary literature supporting this change in recommendation.

In our study documenting an additional 25 cats with lily ingestion, the overall survival rate was 100% (25/25 survived to discharge from the hospital). In this series of cats, 24 of 25 cats were treated with emesis induction, gastric lavage, administration of activated charcoal, IV fluid diuresis, or a combination of these treatments. To evaluate whether length of time that had elapsed since lily ingestion to initiation of treatment had an effect on outcome and the development of AKI, cats were divided into 2 groups: group 1 was defined as lily ingestion ≤ 6 hours prior to initial examination at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania, and group 2 was defined as lily ingestion > 6 up to 48 hours prior to initial examination at the Matthew J. Ryan Veterinary Hospital of the University of Pennsylvania. The group 1 definition was chosen on the basis of the initial abstract from the NAPCC, which suggested that this is the time frame in which gastrointestinal tract decontamination and IV fluid diuresis are effective at eliminating lily-induced nephrotoxicity in cats.[a] In our study, 17 cats were allocated to group 1, and all of these cats had gastrointestinal tract decontamination performed. Sixteen of these 17 cats were hospitalized for IV fluid diuresis, all of which survived to discharge, and only 5 of 16 cats ever had an increase in BUN or creatinine concentration. At the time of discharge from the hospital, only 2 of 16 hospitalized group 1 cats had evidence of ongoing renal dysfunction (a persistent elevation in either BUN or creatinine concentration). These results provide additional clinical evidence supporting the recommendation for early gastrointestinal tract decontamination and IV fluid diuresis in the treatment of lily ingestion. The 1 cat from group 1 that was not hospitalized for IV fluid diuresis had a mildly elevated BUN concentration (35 mg/dL) on initial laboratory testing. Results of follow-up laboratory testing performed 2 days later by the primary veterinarian were normal (BUN concentration, 24 mg/dL; creatinine concentration, 1.3 mg/dL).

We also evaluated the outcome of cats in which a longer period of time had elapsed between ingestion of lily and treatment at our hospital (group 2, > 6 to 48 hours). Interestingly, perhaps the most clinically relevant finding in this study was that 8 of 8 cats allocated to group 2 survived to discharge, including 6 cats evaluated between > 18 and < 48 hours after ingestion. None of the cats in this group had evidence of renal failure at the time of discharge. This is in contrast with the NAPCC abstract,[a] in which there was a mortality rate of 100% in cats treated > 18 hours after ingestion of lily plant. The 1 cat from group 2 that was not hospitalized at our institution was hospitalized at the primary care veterinarian's clinic the following day for 12 to 24 hours. Although we do not have any further laboratory testing results, the owner reported that the cat was doing well approximately 4 years after ingestion of the lily plant material. It must be noted that 5 of the 8 group 2 cats vomited at home prior to initial evaluation, and 2 of these cats likely vomited within the 6-hour window with identifiable lily material seen. This self-decontamination may partially explain the high survival rate found in the present study. Additionally, 2 cats were known to have treatment (including SQ or IV fluids) initiated by the primary care veterinarian prior to referral, which may have also contributed to the high survival rate seen in this group.

The results of this study indicate a good survival rate (100%) for cats that have ingested lily plant within 48 hours prior to treatment. Furthermore, only 7 cats (6 from group 1 and 1 from group 2) had or developed an increase in BUN or creatinine concentration. We suspect that 4 of these cats had prerenal azotemia; 2 had renal azotemia, and in 1 cat, the underlying cause for the azotemia could not be determined from the medical record. Three cats with suspected prerenal azotemia (2 from group 1 and 1 from group 2) had a mild elevation in BUN concentration (33 to 36 mg/dL) and normal creatinine concentration but rapid resolution of the azotemia (all within 3 to 14 hours). Unfortunately, a USG was available for only 1 of these 3 cats with suspected prerenal azotemia. This cat had an elevated USG (> 1.050), supportive of a diagnosis of prerenal azotemia. The fourth cat with suspected prerenal azotemia was the cat from group 1 with a mildly elevated BUN concentration (35 mg/dL) that underwent gastrointestinal tract decontamination and was discharged without further treatment. This cat did not have a USG determined; however, BUN and creatinine concentrations were normal when evaluated 2 days later by the primary care veterinarian.

Of the 2 cats (both in group 1) with suspected AKI, 1 was azotemic (BUN concentration range, 52 to > 100 or 110 mg/dL; creatinine concentration range, 1.6 to 3.1 mg/dL) with inappropriately dilute urine (USG, 1.019) collected 1 hour after admission to the hospital. The second cat suspected to have AKI developed an elevated creatinine concentration while receiving IV fluid therapy. This elevation in creatinine concentration persisted during the course of hospitalization and was still present at the time of the last laboratory testing at 64.5 hours (creatinine concentration, 2.5 mg/dL at this time). Interestingly, this cat had a documented USG of 1.030. However, it was unclear from the medical record what time during hospitalization that this urine was collected, and it may have been prior to the development of sufficient renal injury that would result in a marked decrease in urine concentrating ability.

Finally, the 1 cat (group 1) in which it could not be determined whether the azotemia was renal or prerenal in origin had a single mildly elevated creatinine concentration (2.1 mg/dL) within the first 24 hours of hospitalization. However, this same cat had a normal creatinine concentration (1.7 mg/dL) on initial evaluation and at 25 and 42.5 (1.7 and 1.4 mg/dL, respectively) hours afterward. It is unknown whether this represented a spurious result or a true, transient increase in creatinine concentration. A USG (1.006) for this cat was determined after initiation of IV fluid therapy, impairing interpretation of this result.

At the time of discharge, 3 cats had an elevated BUN or creatinine concentration. Of these 3 cats, 2 were considered to have chronic renal failure on the basis of the presence of persistent azotemia. One (group 1) of these cats was euthanized at a later date (106 days following initial evaluation) for associated problems (a combination of congestive heart failure and chronic renal failure). The other 2 cats were known to be alive approximately 3 and 6 years after initial evaluation. Therefore, long-term

survival is possible, despite the presence of azotemia after lily ingestion.

Treatment recommendations for potential fatal toxicoses, such as lily ingestion, generally include decontamination, treatment with antidotes (if appropriate), and general supportive care. In this study, 19 cats received gastrointestinal tract decontamination procedures for treatment of lily ingestion (all 17 cats from group 1; 2/8 cats from group 2). Emesis induction was the most common gastrointestinal tract decontamination procedure performed (17 cats); however, it had limited efficacy in this series of cats, with a success rate of only 8 of 17. Xylazine was the most commonly used emetic agent, administered to 16 cats, followed by hydrogen peroxide (5 cats), dexmedetomidine (1), and apomorphine (1). In contrast to a previous study[13] that found that xylazine (0.66 mg/kg [0.3 mg/lb], IM) was successful in inducing emesis in 22 of 23 (96%) cats, in our study, xylazine was successful in inducing emesis in only 7 of 16 cats. This may reflect this drug's lack of emetic efficacy or the slightly lower doses used (0.4 to 0.5 mg/kg) or may be secondary to patient and toxicant factors, such as time elapsed since ingestion and the texture and volume of toxicant ingested. Additionally, 4 cats that were administered xylazine had vomited prior to evaluation, and only 1 of these cats was reported to have successful emesis induction. Only 1 cat was administered dexmedetomidine (35.8 µg/kg, IM), and it was successful at inducing emesis. Neither hydrogen peroxide nor apomorphine was documented to be efficacious at inducing emesis in this series of cats.

Gastric lavage as an additional method of gastrointestinal tract decontamination was performed in 4 cats in which emesis induction was unsuccessful. Lily plant material was recovered in the gastric contents of only 1 of these 4 cats. The use of gastric lavage to achieve gastrointestinal tract decontamination is typically reserved for cases in which the toxicant poses a major life-threatening risk to the animal, as is the case with lily ingestion, and when emesis induction is either unsuccessful or contraindicated. Even in cases where emesis induction has been successful, gastric lavage may still be considered if there is concern that there is still a substantial amount of toxicant that has not been recovered (although this was not the case in our study). However, the advantages and disadvantages of this procedure must be considered in each individual case. Some of the risks associated with gastric lavage include the risk associated with anesthesia, the risk of potential aspiration (although this procedure should always be performed with the animal intubated), and the risk of damage to or perforation of the gastrointestinal tract. As with emesis induction, the efficacy of gastric lavage can be variable and is affected by the time between toxicant ingestion and gastric lavage. This may in part explain the low rate of lily plant recovery with gastric lavage in this study. Overall, lily plant material was recovered in 10 of 25 (40%) cats, through self-decontamination (n = 5), extraction from the oropharynx (1), emesis induction (3), or gastric lavage (1).

Interestingly, good outcomes with a low incidence of AKI were found in this series of cats despite the lack of efficacy of emesis induction in the hospital and low recovery rate of actual lily plant material. It is possible that emesis induction or gastric lavage may not be essential in the treatment of cats following lily ingestion and that IV fluid therapy alone may have been responsible for the good outcomes seen in this study. However, this study was not designed to evaluate the effect of different decontamination procedures on outcome with a control group. Therefore, given the low risk associated with emesis induction in otherwise healthy cats, it is our opinion that emesis induction should still be considered in the treatment of lily ingestion in cats.

Although administration of activated charcoal is recommended in cases of lily poisoning and it was administered in 13 cats in the study reported here, it is unknown whether this is an effective treatment. The toxic component in lilies has not been definitively identified, so it is unknown whether it is adsorbed to activated charcoal. In a human toxicology position paper[14] evaluating the results of published studies on activated charcoal administration, the author suggests that a single dose of activated charcoal may be considered when the poison ingestion occurred within the preceding hour (although benefit after this time cannot be ruled out). In addition, the efficacy of activated charcoal is also affected by variable toxicant adsorption, and in some toxicants, it may not provide any additional benefit to the patient. Activated charcoal is not without potential adverse effects, including accidental aspiration, vomiting, and hyperosmolality.[15,16] Future studies on lily toxicosis are needed to determine whether activated charcoal is effective in cases of lily ingestion, the most effective dose, and whether repeated doses of activated charcoal should be administered to protect against enterohepatic recirculation. Until these studies are done, the advantages and disadvantages of administration of activated charcoal should be considered on a case-by-case basis and discussed with the pet owner prior to its administration. The time since lily ingestion, method and ease of administration, and comorbid disease are important considerations prior to administration of activated charcoal in each patient.

Cathartics can be used in combination with activated charcoal not only to increase the rate at which gastrointestinal tract contents are eliminated, therefore reducing the time for toxicant absorption, but also to reduce the potential for desorption of the toxicant from the activated charcoal. The combination of a cathartic with the first dose of activated charcoal continues to be recommended for acute toxicant ingestion in veterinary medicine; however, repeated doses of cathartics are not recommended.[16] In this study, no cats were administered a cathartic as part of the gastrointestinal tract decontamination. Therefore, we cannot comment on the role of cathartics as part of a gastrointestinal tract decontamination plan in the treatment of lily ingestion; however, given the good outcomes seen in this study, it may not be a necessary component.

Twenty-three cats in this study received IV fluid diuresis with crystalloids as treatment for lily ingestion. As the principal effect of lily toxicosis in cats is AKI, the presumption is that IV fluid therapy may aid in minimizing its toxic effects by enhancing diuresis and increasing toxin excretion. The time at which fluid therapy is initiated following ingestion is likely to have an impact on its efficacy. Interestingly, in this study, good outcomes were documented in both groups 1 and 2. Because of the retrospective descriptive design of this study we could not determine whether this result is a direct effect of the gas-

trointestinal tract decontamination and IV fluid diuresis or whether other factors, such as the amount of toxin ingested or other patient factors, played a role in these cats' outcomes. In addition, there were notably fewer cats in group 2, compared with group 1, and therefore, these results should be interpreted cautiously. Although the 2 cats that did not receive IV fluids ultimately did well, these results should be interpreted extremely cautiously owing to the small number, the potentially fatal effects of lily plant ingestion, and the potential for other confounding factors. Future studies are needed to determine the ideal type of fluid used for diuresis as well as the ideal rate and length of administration. This will likely depend on identification of the toxin and determination of its half-life.

Because of its retrospective design, this study has several limitations. First, the exact amount of lily plant ingested by each cat could not be determined, and because the toxic component was unknown, measurement of toxin concentration was not possible. Therefore, it is possible that the good outcomes seen in the cats of the present study may have been due to an inadequate amount of toxin ingested rather than a direct result of decontamination procedures performed. Second, identification of the lily plant and its genus was made by the owner, and in 8 cases, there was no record of the lily type. Therefore, it is possible that in these 8 cats, the lily ingested may not have actually been toxic or the plant ingested may not have been a lily at all. Third, the time at which the lily plant was ingested was also obtained from the owner, and in some circumstances, the time period in which ingestion could have occurred was broad. Although every effort was made to assign cats to the appropriate group on the basis of the time that had elapsed since ingestion, it is possible that some cats could have been assigned to the incorrect group. Additionally, group assignments were determined on the basis of time since ingestion to presentation at our institution, not the referring veterinarian. Some cats may have received initial treatment elsewhere. However, this does not affect the overall survival results or incidence of AKI found in our study. Fourth, treatment protocols among cats were not standardized because of both clinician and owner preference, and therefore information about the efficacy of decontamination procedures should be interpreted cautiously. Fifth, the cats' renal function prior to lily ingestion was unknown, and it is possible that any azotemia seen was not caused by lily ingestion; rather the azotemia may have been secondary to preexisting chronic renal failure. In addition, it is possible that some cases of kidney injury went undetected owing to short hospitalization times. For most cases, long-term follow-up information was also not available, and therefore, assessment of any long-term effects of the lily toxicosis, such as chronic renal failure, was not possible. Sixth, this study was conducted at a hospital that both receives primary cases and serves as a tertiary referral center. Therefore, it is possible that there is bias to this study population and that more seriously ill cats may have been euthanized at their primary care veterinarian and not referred for further care. Finally, there was no control group for this study population. Because of the potentially fatal toxicosis associated with lily ingestion in cats, it is standard of care at our hospital to recommend treatment with both gastrointestinal tract decontamination and IV fluid diuresis. Future studies are needed to determine whether there is any difference in outcome in cats receiving different treatment protocols (ie, gastrointestinal tract decontamination alone, IV fluid diuresis alone, or no treatment at all) following lily ingestion.

The results of this study suggest that the outcome in cats receiving gastrointestinal tract decontamination and IV fluid diuresis within 48 hours after lily ingestion is good. On the basis of the results of this study and literature, we recommend early decontamination after lily ingestion with emesis induction or gastric lavage, followed by activated charcoal administration, IV fluid diuresis, and monitoring of renal parameters. Although we do not know what the cats' outcomes would have been without treatment, or the effects of different decontamination protocols on outcome, given the potential severity of lily toxicosis, we cannot recommend less aggressive treatment at this time. Future studies are needed to determine the most efficacious means of decontamination and the ideal length of IV fluid diuresis in these cats. Additionally, long-term follow-up is needed to determine the incidence of long-term complications associated with lily plant toxicosis in cats.

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