Lily toxicity in cats

Lilies, that is, species of *Lilium* (Liliaceae) (true lily) and *Hemerocallis* (Hemerocallidaceae) (day lily), cause renal damage in cats. All parts of the plant are toxic and a small amount of plant material can have serious consequences if ingested. The mechanism of toxicity remains unknown. Signs develop rapidly with gastrointestinal irritation, followed by polyuria, dehydration and then acute kidney injury. Seizures can occur in severe cases. Treatment is aimed at reducing absorption with emesis and/or activated charcoal and enhancing renal perfusion with intravenous fluid diuresis for at least 48 h. Once acute kidney injury has occurred treatment options are limited and referral should be considered. Prognosis is good in cats where decontamination is prompt and treatment has been started before the onset of renal impairment. Cats with acute kidney injury have a more guarded prognosis.

Lilies, that is, species of *Lilium* (true lily) and *Hemerocallis* (day lily), cause renal failure in cats. Cats are the only species known to develop renal damage from lilies and this was first reported in 1992. Studies and clinical data have shown that these plants do not cause renal toxicity in dogs, rats or rabbits.

It is important to be aware that many plants have lily in their name, such as lily of the valley (*Convallaria majalis*), peace lily (*Spathiphyllum* species) and calla or arum lily (*Zantedeschia aethiopica*). These have different toxic effects; only *Lilium* (Figure 1) and *Hemerocallis* species (Figure 2) are discussed here (Table 1).

**Exposure**

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**Key point**

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Mechanisms of action
The toxic principle(s) and mechanism of lily toxicity are unknown, but renal damage is due to necrosis of renal tubular epithelial cells. The basement membrane remains intact, and prompt, aggressive treatment can result in regeneration of tubular epithelial cells. The mechanism of toxicity involves two processes; initially there is damage to renal tubular epithelium resulting in polyuric renal failure (acute kidney injury) which in turn results in severe dehydration. Whatever the toxic agent, either something in the plant or a feline-specific metabolite, it appears to be eliminated within 48 h; this contention is supported by the observation that fluid diuresis for 48 h can prevent lethal acute kidney injury.

In a small number of cases pancreatic degeneration has been reported and this may be due to a different mechanism.

Toxic dose
All parts of the plant, the pollen, flowers, stem and leaves are toxic. Ingestion of less than one leaf, or part of a flower, could cause severe poisoning. There are also reports of cats developing renal failure after exposure to only pollen (Veterinary Poisons Information Service data).

In an experimental study in cats the

Key point
Ingestion of less than one leaf, part of a flower or just brushing against the plant resulting in pollen exposure can be fatal.
Acute kidney injury

Acute renal failure should now be termed ‘acute kidney injury (AKI)’ to bring veterinary medicine in line with human medicine, and to reflect more accurately the spectrum of pathology occurring in the kidneys, with only a subset of patients with severe disease suffering renal failure. The International Renal Interest Society has useful information on AKI and chronic kidney disease (CKD), the term now used in place of chronic renal failure. See http://www.iris-kidney.com/ for more information.

toxic component was found to be water-soluble, and the aqueous flower extract was more toxic than the aqueous leaf extract. A cat given a dose of 3.84 g of extract (equivalent to eight flowers) died within 4 h.⁹

Clinical signs

Initial signs after ingestion of lilies are due to gastrointestinal irritation and usually start within 1–6 h, whereas the later effects are due to uraemia.

Clinical signs first observed may include hypersalivation, vomiting, anorexia, weakness, lethargy and depression. The vomiting usually resolves within 12 h, but recurs again at 36–48 h due to uraemia.²

There is polyuria from around 12–30 h, followed by dehydration at 18–30 h. As acute kidney injury develops owners may note depression, weakness, anorexia and vomiting.¹⁰ Physical examination will show signs typical of acute kidney injury such as dehydration, oral ulceration, uraemic breath, enlarged, painful kidneys and reduced urine production (anuria).¹⁰ Seizures can occur in cats with severe acute kidney injury.⁹

Laboratory findings

The main finding in cats with lily exposure is severe azotaemia. Biochemical changes with rising urea, creatinine, potassium and phosphorus concentrations generally occur from 18–24 h.⁸ The creatinine may be disproportionally elevated compared to the urea.¹⁰,¹¹ There is tubular necrosis with haematuria, proteinuria, glycosuria, isosthenuria, squamous and epithelial cells in the urine and numerous urine casts.

A mild elevation in liver enzymes may be noted but may be due to severe anorexia and hepatic stress.⁷ Pancreatitis has been reported in a few cases.⁹,¹²

Prognosis

Prognosis is good in cats where decontamination is prompt and treatment has been started before the onset of renal impairment. It is generally stated that treatment started more than 18 h after ingestion is associated with a poor outcome,⁵ however, a recent analysis of 25 cases found that outcome was good in cats treated with gastrointestinal decontamination, intravenous fluid diuresis or both within 48 h of ingestion. It is worth noting however, that in this study most cats (68%) presented within 6 h and only eight (32%) presented after more than 6 h. Of these, five

Tip

Prompt treatment with gastric decontamination and fluid diuresis is associated with a good outcome so any cat with suspected or confirmed exposure should be seen and treated immediately. Cats treated later and cats with acute kidney injury have a guarded prognosis.
had vomited at home and in two cases it was likely to have occurred within 6 h of ingestion.\(^4\)

In a review of 55 cases of feline lily exposure with known outcome, 48 cats (87\%) had either brief clinical signs or remained asymptomatic. Three cats (5.5\%) had continuing abnormalities in renal parameters, one (1.8\%) had aspiration pneumonia and three cats (5.5\%) were euthanased with renal failure. In this group of 55 cats, six (11\%) developed renal signs.\(^3\)

Cats with acute kidney injury have a guarded prognosis and those with anuria have a grave prognosis.\(^10\) In cats given no treatment death can occur 3–7 days after ingestion of lilies.\(^2,8\) In cats that survive chronic kidney disease may occur as a long-term consequence of lily nephrotoxicity.\(^10\)

**Treatment**
The aim of treatment is to reduce absorption and support renal function. If exposure to lilies is suspected advice should be sought from the VPIS and a veterinary specialist, with prompt referral to a specialist centre considered.

**Decontamination**
An emetic and/or activated charcoal can be given if ingestion was recent. If vomiting has already occurred an antiemetic and activated charcoal should be given. It is important to wash the cat thoroughly to remove any residual pollen on the coat, face or feet.

**Tip**
Wash exposed cats thoroughly to remove any residual pollen on the coat or feet but monitor temperature to avoid hypothermia.

**Monitoring**
Monitoring of renal function is essential. The hydration status should also be monitored by calculating fluid in and fluid out, which allows appropriate adjustment of the fluid rate as required. Placement of a urinary catheter is important to allow accurate monitoring of urine output. Urine analysis will show evidence of renal tubular injury (eg, renal casts, isothenuria, glucosuria) before the onset of azotaemia,\(^11\) and these changes can be observed as early as 12 h after ingestion.\(^7,8\)

**Prevention of renal injury**
Treatment in cats with lily exposure is aimed at preventing anuria and enhancing renal perfusion. Fluid rates (Figure 3) should be based on percentage dehydration at presentation, ongoing losses (via polyuria and vomiting for example) and adequate to enhance renal perfusion without volume overload. Monitoring blood pressure, bodyweight and urine output is recommended, with measurement of central venous pressure ideal but often unavailable. The fluid should be either isotonic saline or a polyionic solution such as Hartmann’s solution.\(^10\) In all cats,
particularly those with reduced urine production or cardiomyopathy, volume overload should be avoided with close monitoring (see Fluid therapy in cats 1 and 2 Feline Focus 2015; 1(1): 7–12 and 2015; 1(2): 41–47). Rehydration and restoration of renal perfusion and urine output usually corrects metabolic abnormalities, but severe hyperkalaemia should be treated appropriately (dextrose, insulin).10

Management of acute kidney injury
Once renal damage has occurred treatment options are limited. Although haemodialysis and peritoneal dialysis have been successful in some cases,6,7,13 availability may be limited. Other drugs commonly used in the management of acute kidney injury, such as furosemide or mannitol, are not very effective in cats with lily-induced renal toxicity.11,12 As mentioned previously, consulting a veterinary specialist for advice on treatment, or referral is strongly recommended.

Conclusions
Cats appear to be uniquely sensitive to lilies (Figure 4) and develop acute renal injury following exposure. Successful treatment relies on prompt decontamination and fluid diuresis to maintain renal function. Once renal failure occurs treatment options are limited and there is a risk

of chronic kidney disease. Raising awareness and prevention are therefore key to reducing cases of lily-induced renal failure in cats.

References