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CASE REPORT

Case Report: Lily (*Lilium Orientalis*) Poisoning in a Cat

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ABSTRACT

Some lily species are toxic to cats. The main harmful effect is on the kidneys and leads to acute renal damage. Ingestion of any part of the plant can cause poisoning. Even the ingestion of small amounts of plant parts can have serious consequences. The mechanism of toxicity is not known exactly. Symptoms develop rapidly. In the progress of the disease, the gastrointestinal system is primarily affected. Afterward, polyuria, dehydration, and renal failure accompany the symptoms. Seizures may occur in severe cases. In acute cases, the animal may be tried to induce vomiting, and/or applications to reduce toxin absorption may be made. Renal perfusion is attempted to be provided with intravenous fluid applications. Treatment options are more limited in the cases of lily intoxication if renal failure develops. Taking precautions against the plant is more effective than treatment. Therefore, it is important to raise awareness of cat owners. In the present case, stargazer poisoning in an elderly female British Shorthair cat brought to a private veterinary clinic for examination and treatment was discussed. Toxication was diagnosed based on clinical, hematological, and biochemical findings. The cat presented with symptoms such as vomiting, anorexia, lethargy, and urinary obstruction, all indicative of lily toxicosis. This case report aims to emphasize the toxicity that may be caused by lily plants in cats living at home. Also, it provides information about diagnostic and therapeutic procedures.

Key Words: Feline, Nephrotoxicity, Plant, Toxication, Toxicity

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Vaka Raporu: Bir Kedide Zambak (*Lilium Orientalis*) Zehirlenmesi

ÖZ

Bazı zambak türleri kediler için toksiktir. Başlıca toksik etkisi böbrekler üzerinde görülür ve akut renal hasara yol açar. Bitkinin herhangi bir kısmının alınması zehirlenmeye neden olabilir. Az miktarda bitki parçası yutulduğunda dahi ciddi sonuçlarla karşılaşılabilir. Toksisitenin mekanizması tam olarak bilinmemektedir. Belirtiler hızla gelişir. Hastalık seyrinde öncelikle gastrointestinal sistem etkilenir, sonrasında tabloya poliüri, dehidrasyon ve böbrek yetmezliği eşlik eder. Şiddetli vakalarda nöbet görülebilir. Akut olgularda hayvan kusturulmaya çalışılabilir ve/veya toksin emilimini azaltıcı uygulamalar yapılabilir. İntravenöz sıvı uygulamaları ile renal perfüzyon sağlanmaya çalışılır. Böbrek yetmezliği gelişmiş toksikasyon vakalarında tedavi seçenekleri daha sınırlıdır. Bitkiye karşı önlem almak tedaviden daha etkilidir. Bu nedenle kedi sahiplerinin bilinçlendirilmesi önemlidir. Sunulan raporda özel bir veteriner kliniğe muayene ve tedavi amaçlı getirilen bir yaşlı dişi british shorthair ırkı kedide stargazer zehirlenmesi konu edildi. Anamnezde kusma, iştahsızlık, durgunluk ve idrar yapamama şikayeti bulunan kediye klinik, hematolojik ve biyokimyasal bulgular ve anamnez bilgi dahilinde zambak toksikasyonu tanısı kondu. Bu olgu sunumu; evde yaşayan kedilerde zambak bitkisinin neden olabileceği toksikasyona dikkat çekmeyi ve yanı sıra tanı ve tedavi prosedürü hakkında bilgi vermeyi hedeflemektedir.

Anahtar Kelimeler: Bitki, Kedi, Nefrotoksisite, Toksikasyon, Toksisite

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INTRODUCTION

Cases of plant toxicity in cats and dogs are common because most of them live at home and have occasional access to streets, parks, and rural areas. Ornamental plants found in gardens and widely used for home decoration are responsible for many of the poisonings (Andrade, 2011).

Although there are many lily species, two of them cause significant nephrotoxicity in cats. These are *True Lilies (Lilium ssp.) and Day Lilies (Hemerocallis ssp.)*. Even the ingestion of a small plant part can be toxic to cats. On the other hand, some species are not toxic (Fitzgerald, 2010). The exact mechanism of action of lily-induced nephrotoxicity is unknown. However, the rapid onset of clinical signs after ingestion of the poisonous species indicates a rapid rate of absorption (Hall, 2006). The lily-induced nephrotoxic syndrome has only been observed in cats. No age, sex or breed-related predisposition has been identified. Rats, mice, and rabbits consume up to 1.5 times their body weight in lilies, yet no nephrotoxic damage is observed (Hall, 2007). On the other hand, similarly, lily does not produce nephrotoxic effects even if consumed in large amounts by dogs. Only vomiting and gastrointestinal symptoms are observed (Fitzgerald, 2010).

Clinical signs of lily poisoning in cats include hypersalivation, vomiting, anorexia, lethargy, polyuria, polydipsia, azotemia, glucosuria, proteinuria, and in severe cases oliguria/anuria (Rumbeiha et al., 2004). The expected course of poisoning is as follows: 1-3 hours after ingestion lily causes decreased appetite, hypersalivation, vomiting, and depression in cats. Vomiting and hypersalivation may persist for 2 to 6 hours. Gastrointestinal signs subside after about 6 hours. At 12-30 hours, polydipsia and polyuria are observed due to progression of kidney damage. This is followed by dehydration which further increases the effects of kidney damage. Within 24 to 48 hours, the condition progresses to an oliguric phase, followed by an anuric phase. When renal activity stops, metabolic wastes accumulate in the body and cause the resumption of vomiting. Asthenia usually develops 30-72 hours after lily consumption. In 3-7 days, the symptoms gradually worsen and the case ends in death (Fitzgerald, 2010). Apart from the nephrotoxic effect, pancreatic damage and seizures may also be observed (Rumbeiha et al., 2004).

There is no antidote or specific treatment for this intoxication. If the condition is recognised early, supportive treatment can be given to manage symptoms and provide a chance of recovery. Successful treatment involves initiating fluid diuresis before the onset of anuric renal failure (Bates, 2006). Since drugs such as furosemide and mannitol used to stimulate urine output when anuria develops are not very successful in treatment, peritoneal dialysis or hemodialysis is thought to be the only potential treatment (Tefft, 2004; Hall, 2013). However, in one case, oligo-anuric acute kidney injury after lily toxicity was reported to be resolved by medical management (To et al., 2023).

CASE HISTORY

The material of this study was a 1-year-old female British Shorthair cat. In the anamnesis, it was learned that the cat had eaten a house plant (lily) approximately 21 hours before the first clinic application and had been taken to a veterinary clinic (first clinic) with the complaint of vomiting. It was also learned that; at this initial clinic, the patient was evaluated clinically, hemogram and biochemistry analyses as well as radiographically and was treated in accordance with the following protocol. The results of the hemogram and biochemical analyses associated with this initial presentation are given in Tables 1 and 2. According to the data provided by the history; intravenous balanced solution, H2-receptor antagonist, antihistamine, choleretic and amino acid solution, as well as antiemetic and nonsteroidal anti-inflammatory was applied at the first clinic. After the treatment, the vomiting stopped and the patient was discharged. The cat, which symptoms reappeared about 12 hours later, was brought to another veterinary clinic (*Florya Doğa Veterinary Clinic, İstanbul, Türkiye*) approximately 24 hours after being discharged from the first clinic with complaints of anuria, loss of appetite, difficulty breathing, and recurrent vomiting. Blood analyses (Tables 3 and 4) and radiographic examination were repeated immediately. The patient was evaluated ultrasonographically. Emergency dialysis was considered for the patient whose treatment was started with slow fluid replacement subcutaneously. However, due to the delay in the case, our patient, whose general condition was quite poor and whose respiration was depressed, died after a short time despite intubation.

Ultrasonographic examination revealed bilateral perinephric fluid collection (Figure 1). Resistive index measurements were 0.79 for the right kidney and 0.79 for the left kidney (Figure 2). The empty urinary bladder could not be evaluated. Radiography revealed ascites in the abdomen as well as pulmonary edema (Figure 3). Biochemical analyses demonstrated elevated levels of creatinine, ALT, BUN, glucose, calcium, phosphorus, and total bilirubin, while chloride, sodium, and total protein levels were found to be decreased (Table 3). Hemogram results showed that RBC, HGB, MCHC, and neutrophil values were higher than references (Table 4).

Table 1. Hematological findings (initial clinical results)

Parameter	Result	Reference Range (Fielder, 2024)	Unit
WBC	9.15	5.5-19.5	$x10^9/L$
Neutrophils	7.99	1.8-12.6	$x10^9/L$
Neutrophils	87.3	45.0-64.0	%
Eosinophils	0.38	0.0-0.8	$x10^9/L$
Eosinophils	4.2	0.0-4.0	%
Lymphocytes	0.47	1.5-7.0	$x10^9/L$
Lymphocytes	5.1	27.0-36.0	%
Monocytes	0.31	0.0-0.9	$x10^9/L$
Monocytes	3.4	0.0-5.0	%
RBC	11.28	5.0-10.0	$x10^{12}/L$
Hemoglobin	17.0	9.8-15.4	g/dL
MCV	40.5	39.0-55.0	fL
MCH	15.1	13.0-17.0	pg
MCHC	37.3	30.0-36.0	g/dL
Hematocrit	45.7	26.0-51.0	%
Platelets	427	100.0-518.0	$x10^9/L$
MPV	11.0	12-18	fL

*WBC, White Blood Cell count; RBC, Red Blood Cell count; MCV, Mean Corpuscular Volume; MCH – Mean Corpuscular Hemoglobin; MCHC, Mean Corpuscular Hemoglobin Concentration; MPV, Mean Platelet Volume

Table 2. Biochemistry measurement data (initial clinical results)

Parameter	Result	Reference Range (Fielder, 2024)	Unit
ALP	28	0-45	U/L
ALT	482	25-97	U/L
BUN	111.6	19-34	mg/dL
BUN/CRE	11.4	4.0-33.0	mg/dL
CRE	9.83	0.9-2.2	mg/dL
GLU	132	60.0-120.0	mg/dL
TP	6.7	6.0-7.9	g/dL

*ALP, Alkaline Phosphatase; ALT, Alanine Aminotransferase; BUN, Blood Urea Nitrogen; BUN/CRE, Blood Urea Nitrogen / Creatinine Ratio; CRE, Creatinine; GLU, Glucose; TP, Total Protein

Table 3. Biochemistry measurement data (the data from the second clinic)

Parameter	Result	Reference Range (Fielder, 2024)	Unit
Cl^-	94.00	115.00 - 130.00	mEq/L
K^+	5.30	3.40 - 4.60	mEq/L
Na^+	127.00	146.00 - 156.00	mEq/L
Na/K	23.96	0.00-0.00	mEq/L
CRE	15.33	0.9-2.2	mg/dL
ALB	2.50	2.80 - 3.90	g/dL
ALB/GLB	0.83	0.35 - 1.50	g/dL
ALP	40.00	0.00 - 45.00	U/L
ALT	208.00	25.00 - 97.00	U/L
GGT	10.00	1.00 - 10.00	U/L
GLU	169.00	60.00 - 120.00	mg/dL
BUN	140.00	19.0-34.0	mg/dL
BUN/CRE	9.13	4.00 - 33.00	mg/dL
Ca^{++}	12.00	8.70 - 11.70	mEq/L
IP	15.00	3.0 - 6.10	mEq/L
TBIL	1.10	0.0 - 0.10	mg/dL
TCHO	133.00	71.0-156.0	mg/dL
TP	5.50	6.0 - 7.90	g/dL

*Cl^-, Chloride; K^+, Potassium; Na^+, Sodium; Na/K, Sodium/Potassium Ratio; CRE, Creatinine; ALB, Albumin; ALB/GLB, Albumin/Globulin Ratio; ALP, Alkaline Phosphatase; ALT, Alanine Aminotransferase; GGT, Gamma-Glutamyl Transferase; GLU, Glucose; BUN, Blood Urea Nitrogen; BUN/CRE, Blood Urea Nitrogen/Creatinine Ratio; Ca^{2+}, Ionized Calcium; IP, Inorganic Phosphorus; TBIL, Total Bilirubin; TCHO, Total Cholesterol; TP, Total Protein

Table 4. Hematological findings (the data from the second clinic)

Parameter	Result	Reference Range (Fielder, 2024)	Unit
WBC	14.33	5.5-19.5	$x10^9/L$
Neutrophil	11.65	1.8-12.6	$x10^9/L$
Neutrophil	81.30	45.0-64.0	%
Eosinophil	0.46	0.0-0.8	$x10^9/L$
Eosinophil	3.20	0.0-4.0	%
Lymphocyte	1.63	1.5-7.0	$x10^9/L$
Lymphocyte	11.40	27.0-36.0	%
Monocyte	0.44	0.0-0.9	$x10^9/L$
Monocyte	3.10	0.0-5.0	%
RBC	11.93	5.0-10.0	$x10^{12}/L$
Hemoglobin	16.90	9.8-15.4	g/dL
MCV	36.80	39.0-55.0	fL
MCH	14.20	13.0-17.0	pg
MCHC	38.50	30.0-36.0	g/dL
Hematocrit	43.90	26.0-51.0	%
Platelets	377.00	100.0-518.0	$x10^9/L$
MPV	10.80	12-18	fL

*WBC, White Blood Cell count; RBC, Red Blood Cell count; MCV, Mean Corpuscular Volume; MCH – Mean Corpuscular Hemoglobin; MCHC, Mean Corpuscular Hemoglobin Concentration; MPV, Mean Platelet Volume

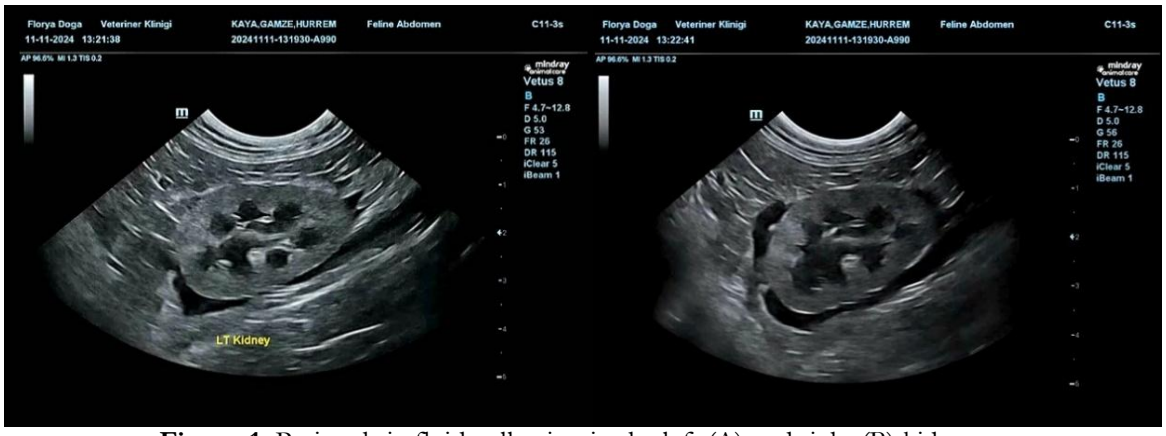

Figure 1: Perinephric fluid collection in the left (A) and right (B) kidneys.

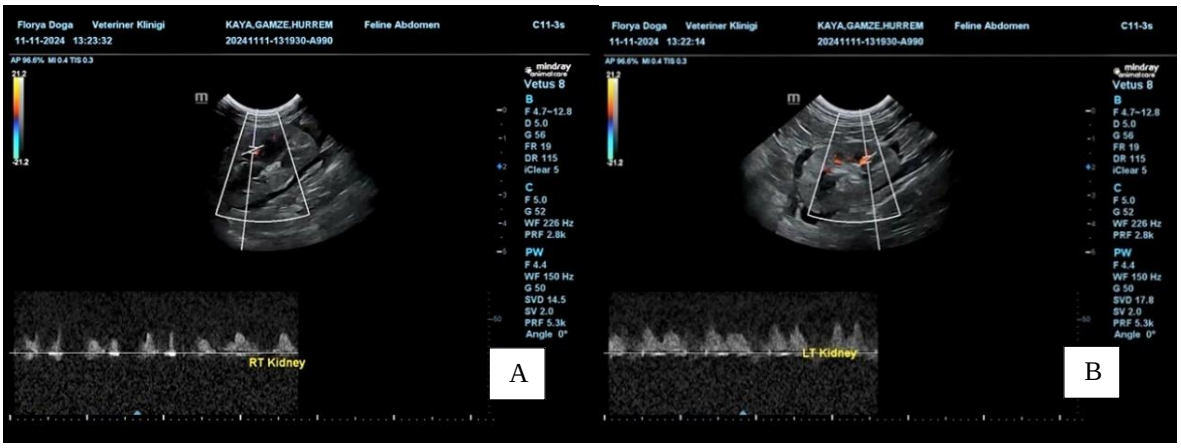

Figure 2: Resistive index measurement in the right (A) and left (B) kidneys.

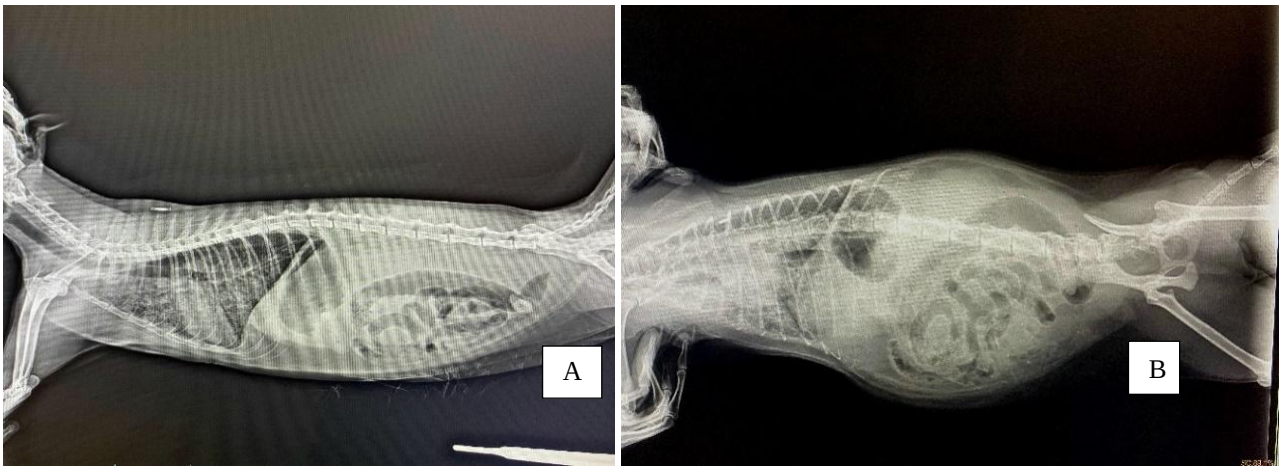

Figure 3: Appearance consistent with pulmonary edema (A) and ascites (B) on radiographs.

DISCUSSION

Cases of plant-based poisoning in cats are frequently encountered in domestic environments. Especially Lilium spp. and Hemerocallis spp. are widely used decorative plants in homes and gardens. All parts of the lily plant are dangerous for cats. Symptoms of toxicity occur rapidly after ingestion (Fitzgerald, 2010). These symptoms include vomiting, lethargy, loss of appetite, and polydipsia. The clinical findings observed in the present case are similar to those reported in the literature. In this case, considering the patient history and the time spent in the first clinic, it was evaluated that anuria probably developed between 40 and 50 hours after ingestion of the plant. Again; BUN, creatinine as well as potassium levels, which are used to determine the current degree of renal effects, were observed to be increased in our case. Creatinine elevation in cats is primarily related to glomerular filtration rate, commonly due to kidney injury, dehydration, or urethral obstruction. In lily toxicosis, marked creatinine rise secondary to acute tubular necrosis, which results in severe renal dysfunction (Hall, 2007). The serum creatinine level, which is the most important parameter that helps to determine lily intoxication, increased independently of the BUN level in this case (Hall, 2004). In the ultrasonographic examination, the resistive index value for both kidneys was 0.79. In cats, values of 0.70 and above are considered pathological (Debruyn et al., 2012). An increased index indicates an increase in vascular resistance similar to renal arterial stenosis and a marked renal disease (Granata et al., 2009). However; ALT activity has the highest sensitivity (more than 80%) for hepatic disorders in cats (Kozat and Sepehrizadeh, 2017). Cats are generally predisposed to develop hepatobiliary pathologies associated with stress and weight loss (Oikonomidis and Milne, 2023). It was evaluated that marked lethargy and hepatic stress may be related to the increase in ALT serum level (Hall, 2013) in this case.
Pulmonary edema, which has been reported in some cases, was also observed in our case (Langston, 2002; Fitzgerald, 2010).
In the presented case, it was evaluated that the treatment applied to the patient at first clinical application and the cessation of vomiting could possibly be related to the fact that the applied treatment was probably more symptomatic and coincided with the beginning and middle stages of the case. This cat, which complaints recurred after a while and entered the anuric phase, probably already had acute renal failure before applying to another clinic for the second time. However, despite these efforts, the patient died. In contrast to the present case, Langston (2002) reported that three out of six cats survived following lily toxication. However, all of these survivors developed chronic kidney disease and required long-term management, including renal-supportive diets such as protein-restricted formulations. The remaining three cats in that study either died or were euthanized.

CONCLUSION

In conclusion, lily poisoning is an extremely critical and life-threatening condition for cats, which must be clinically acknowledged and urgently treated. For the successful management of similar cases, it is of utmost importance for cat owners to provide accurate and timely history to the veterinarian and for veterinary clinics to be knowledgeable about the condition. There is currently no specific or definitive treatment for this toxicity, which leads to a poor prognosis.
The key to the successful treatment of lily toxicity is the preservation of renal function. For this purpose, rapid decontamination and fluid diuresis should be initiated. Early intervention results in a better prognosis. However, in delayed treatment, the chances of recovery are minimal. Prevention is the most effective strategy. To minimize risk, lilies should be kept out of reach in households with cats, or ideally, lilies should not be present at all. Cat owners should prefer non-toxic plants in their living environments, which would be the most appropriate approach.

Conflict of interest: The authors have no conflicts of interest to report.

Authors' Contributions: TC and ZB contributed to the project idea, design and execution of the study. SNK and EE contributed to the acquisition of data. TC analysed the data. ZB and SNK drafted and wrote the manuscript. TC, ZB and SNK reviewed the manuscript critically. All authors have read and approved the finalized manuscript.

Ethics Committee Information: This study is not subject to the permission of HADYEK in accordance with the "Regulation on Working Procedures and Principles of Animal Experiments Ethics Committees" 8 (k). The data, information and documents presented in this article were obtained within the framework of academic and ethical rules.

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