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Acute Kidney Injury Following Lily Toxicity in a Domestic Cat: Diagnosis and Clinical Management

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Lily toxicity is one of the most severe and life-threatening plant intoxications encountered in feline medicine, with ingestion of even a small quantity of plant material capable of inducing acute kidney injury (AKI). Members of the genera *Lilium* and *Hemerocallis*, including Easter lily (*Lilium longiflorum*), Tiger lily (*Lilium lancifolium*), Asiatic lily (*Lilium asiaticum*), Japanese show lily (*Lilium speciosum*), Stargazer lily (*Lilium orientalis*), and Daylily (*Hemerocallis* spp.), are highly nephrotoxic to cats. Although the precise nephrotoxic principle remains unidentified, rapid absorption following ingestion results in selective injury to renal proximal tubular epithelial cells, culminating in acute tubular necrosis and severe impairment of renal function. Clinical signs typically develop within hours of exposure and rapidly progress from gastrointestinal disturbances to oliguric or anuric renal failure if treatment is delayed. Prompt diagnosis based on exposure history, clinical presentation, laboratory evaluation, urinalysis, and diagnostic imaging is critical because early aggressive fluid therapy initiated within 18 hours of ingestion markedly improves prognosis. Echogenic renal changes identified by ultrasonography, together with biochemical evidence of azotemia and electrolyte disturbances, support diagnosis and therapeutic monitoring. This review summarizes the pathophysiology, clinical manifestations, diagnostic approaches, current treatment strategies, prognostic indicators, and recent advances in the management of lily-induced acute kidney injury in domestic cats.

Introduction

Acute kidney injury (AKI) is a common emergency encountered in feline clinical practice and represents a syndrome characterized by the abrupt decline in renal function, resulting in impaired glomerular filtration, accumulation of nitrogenous waste products, electrolyte imbalance, acid–base disturbances, and fluid dysregulation. Among toxic causes of AKI in cats, lily intoxication remains one of the most devastating because of its rapid progression and exceptionally high nephrotoxic potential. Unlike many toxic plants that produce mild gastrointestinal irritation, true lilies cause selective and severe renal tubular damage in cats, even after ingestion of minute quantities. Cats are uniquely susceptible to lily toxicity. Consumption of only one or two petals, a single leaf, pollen adhering to the fur, or even water from a vase containing lilies may produce fatal renal injury. Dogs and most other domestic species generally experience only mild gastrointestinal upset following lily ingestion, emphasizing the remarkable species-specific susceptibility of felines. The widespread use of lilies as ornamental garden plants, floral decorations, and holiday gifts has increased the frequency of accidental feline exposure. Consequently, lily intoxication constitutes a true veterinary emergency requiring immediate recognition and aggressive intervention. Delays

exceeding 18–24 hours following ingestion substantially reduce the likelihood of renal recovery and markedly increase mortality.

Etiology and Toxic Plants

True lilies belonging to the family Liliaceae and certain members of the family Asphodelaceae possess nephrotoxic properties in cats. The most commonly implicated species include Easter lily (*Lilium longiflorum*), Tiger lily (*Lilium lancifolium*), Asiatic lily (*Lilium asiaticum*), Stargazer lily (*Lilium orientalis*), Japanese show lily (*Lilium speciosum*), Rubrum lily (*Lilium speciosum var. rubrum*), and Daylily (*Hemerocallis* spp.). Every component of these plants—including flowers, petals, leaves, stems, pollen, bulbs, and vase water—contains sufficient toxin to induce renal injury. Although numerous investigations have attempted to identify the toxic principle, the nephrotoxic compound remains unknown. Current evidence suggests that the toxin is rapidly absorbed from the gastrointestinal tract and selectively accumulates within proximal renal tubular epithelial cells, initiating irreversible cellular injury.

Pathophysiology

Following ingestion, the nephrotoxic compound is rapidly absorbed through the gastrointestinal tract and distributed via systemic circulation. Within the kidneys, the toxin preferentially targets proximal convoluted tubular epithelial cells where it induces mitochondrial dysfunction, oxidative stress, disruption of cellular metabolism, and apoptosis followed by coagulative necrosis. Loss of tubular epithelial integrity results in impaired reabsorption of water, glucose, amino acids, bicarbonate, sodium, and electrolytes. Progressive tubular necrosis reduces glomerular filtration rate, promotes intratubular obstruction by necrotic cellular debris, and activates inflammatory pathways that further exacerbate renal injury. Initially, affected cats develop polyuria due to impaired tubular concentrating ability. As renal injury progresses, urine production decreases, culminating in oliguria or anuria. Progressive accumulation of uremic toxins leads to systemic manifestations including metabolic acidosis, hyperkalemia, hypertension, gastrointestinal ulceration, encephalopathy, and cardiovascular instability. Histopathological examination consistently demonstrates diffuse degeneration and necrosis of proximal tubular epithelial cells with intraluminal casts, tubular dilation, interstitial edema, and variable inflammatory infiltration.

Clinical Presentation

Clinical signs develop rapidly and generally follow a predictable chronological sequence. Within the first one to three hours following ingestion, affected cats commonly exhibit hypersalivation, vomiting, anorexia, depression, lethargy, and occasionally abdominal discomfort. During the next six to twelve hours, gastrointestinal signs persist while dehydration progressively develops because of vomiting and polyuria. Between twelve and twenty-four hours after exposure, renal injury becomes clinically evident. Cats demonstrate worsening lethargy, dehydration, polyuria, polydipsia, and reduced appetite. Serum creatinine and blood urea nitrogen concentrations begin to increase during this period. By twenty-four to seventy-two hours, severe acute kidney injury becomes established. Oliguria or anuria develops in many patients, accompanied by marked azotemia, oral ulceration, halitosis, hypothermia, bradycardia secondary to hyperkalemia, neurologic depression, seizures, and eventually coma if untreated.

Diagnostic Approach

Diagnosis relies upon integration of exposure history, clinical findings, laboratory evaluation, urinalysis, diagnostic imaging, and exclusion of alternative causes of acute kidney injury. Obtaining an accurate history remains critically important. Owners may report chewing on lily flowers, grooming pollen from the coat, ingestion of leaves, or access to bouquets containing lilies. Because many owners are unaware of lily toxicity, identification of the plant species may require photographs or botanical confirmation. Physical examination commonly

reveals dehydration, depression, vomiting, painful kidneys on abdominal palpation, oral ulceration, uremic breath, and altered urine output depending upon disease stage.

Hematological and Biochemical Findings

Complete blood count findings are frequently nonspecific but may reveal hemoconcentration resulting from dehydration and mild leukocytosis associated with stress or inflammation. Serum biochemical abnormalities include rapidly increasing blood urea nitrogen and creatinine concentrations, hyperphosphatemia, hyperkalemia during oliguric stages, metabolic acidosis, and variable hypocalcemia. Elevated symmetric dimethylarginine (SDMA) concentrations may identify renal dysfunction earlier than creatinine in some patients. Serial biochemical monitoring is essential because renal injury may continue to progress during the first forty-eight to seventy-two hours despite initiation of therapy.

Urinalysis

Urinalysis frequently detects early renal tubular dysfunction before severe azotemia develops. Common findings include isosthenuria or inadequately concentrated urine, glucosuria in the absence of hyperglycemia, proteinuria, granular casts, epithelial casts, renal tubular epithelial cells, mild hematuria, and enzymuria reflecting tubular injury. Measurement of urinary biomarkers such as neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), cystatin C, and urinary gamma-glutamyl transferase may permit earlier detection of tubular injury, although routine clinical availability remains limited.

Diagnostic Imaging

Abdominal ultrasonography provides valuable information regarding renal morphology and assists exclusion of obstructive uropathy or congenital renal abnormalities. Early ultrasonographic findings include bilateral renomegaly, increased cortical echogenicity, loss of corticomedullary distinction, hyperechoic renal cortex, mild pyelectasia, and perirenal edema. As disease progresses, kidneys may become swollen with diffuse parenchymal hyperechogenicity reflecting widespread tubular necrosis. Thoracic radiography may be indicated in severely affected cats to evaluate pulmonary edema associated with fluid overload during aggressive intravenous fluid therapy.

Differential Diagnosis

Lily toxicity should be differentiated from other causes of feline acute kidney injury, including ethylene glycol intoxication, nonsteroidal anti-inflammatory drug toxicity, aminoglycoside nephrotoxicity, leptospirosis, pyelonephritis, ureteral obstruction, urethral obstruction, renal lymphoma, septicemia, severe dehydration, and ischemic acute tubular necrosis. A confirmed history of lily exposure combined with characteristic clinical progression strongly supports diagnosis.

Clinical Management

Successful treatment depends primarily upon the interval between toxin exposure and initiation of therapy. Cats presented within six to eighteen hours following ingestion have a substantially better prognosis than those treated after development of established renal failure. If ingestion occurred within two hours and the patient is neurologically normal, emesis may be induced under veterinary supervision. Administration of activated charcoal may reduce gastrointestinal absorption, although evidence regarding efficacy remains limited because of the rapid absorption of the nephrotoxin. Aggressive intravenous isotonic crystalloid fluid therapy represents the cornerstone of treatment. Fluids should be administered for at least forty-eight to seventy-two hours to maintain renal perfusion, enhance toxin elimination, and prevent progression of tubular injury. Urine output, hydration status, body weight, blood pressure, and central venous pressure should be monitored to avoid fluid overload. Antiemetics such as maropitant, ondansetron, or dolasetron improve patient comfort and facilitate nutritional support. Gastroprotectants may be indicated in cats exhibiting severe vomiting or uremic gastritis. Nutritional support should be instituted early

because prolonged anorexia predisposes cats to hepatic lipidosis. Electrolyte abnormalities require individualized correction. Hyperkalemia should be treated promptly using intravenous calcium gluconate for myocardial stabilization, insulin combined with dextrose, sodium bicarbonate when metabolic acidosis is severe, or beta-adrenergic agonists where appropriate. Cats developing oliguric or anuric acute kidney injury despite aggressive medical management may require renal replacement therapy. Intermittent hemodialysis and continuous renal replacement therapy effectively remove uremic toxins while allowing recovery of damaged renal tissue. Peritoneal dialysis represents an alternative where hemodialysis is unavailable.

Monitoring During Hospitalization

Continuous monitoring is essential throughout hospitalization. Serial assessment should include urine output measurement, body weight, hydration status, serum creatinine, blood urea nitrogen, phosphorus, potassium, acid–base status, blood pressure, packed cell volume, and total protein. Electrocardiographic monitoring is recommended in patients with significant electrolyte disturbances or suspected cardiac arrhythmias. Repeated ultrasonographic examination may assist evaluation of renal morphology and detection of complications such as hydronephrosis or progressive renal swelling

Prognosis

Prognosis depends primarily upon the timing of treatment and severity of renal injury at presentation. Cats receiving aggressive decontamination and intravenous fluid therapy within eighteen hours of exposure often recover completely without permanent renal dysfunction. Development of azotemia significantly worsens prognosis, while oliguric or anuric renal failure carries a guarded to poor prognosis unless renal replacement therapy is available. Long-term survivors generally regain satisfactory renal function, although some may develop chronic kidney disease secondary to incomplete tubular regeneration and residual nephron loss.

Prevention

Because there is no specific antidote for lily toxicity, prevention remains the most effective strategy. Cat owners should avoid keeping true lilies or daylilies in homes containing cats. Veterinary professionals should educate owners regarding the extreme nephrotoxicity of lilies and encourage immediate veterinary evaluation following any suspected exposure, regardless of the amount ingested or the absence of clinical signs. Public awareness campaigns involving florists, garden centers, pet stores, and veterinary clinics can substantially reduce accidental feline exposure.

Future Perspectives

Continued research is focused on identifying the unknown nephrotoxic compound responsible for lily-induced renal injury. Advances in metabolomics, proteomics, and toxicogenomics may facilitate characterization of the toxic principle and enable development of targeted antidotal therapies. Novel renal biomarkers, including NGAL, KIM-1, liver-type fatty acid-binding protein (L-FABP), clusterin, and microRNA profiles, may permit earlier diagnosis before conventional biochemical abnormalities become apparent. Artificial intelligence-assisted clinical prediction models integrating laboratory data, imaging findings, and biomarker profiles may improve prognostic accuracy and optimize individualized treatment strategies. Emerging regenerative therapies involving mesenchymal stem cells, extracellular vesicles, and renal tissue engineering hold promise for enhancing tubular regeneration and improving recovery following severe acute kidney injury.

Conclusion

Lily toxicity represents one of the most serious toxicological emergencies in feline medicine because of its capacity to induce rapid and often irreversible acute kidney injury. Early recognition of exposure, prompt gastrointestinal decontamination when appropriate,

aggressive intravenous fluid therapy, intensive monitoring, and timely referral for renal replacement therapy are fundamental determinants of survival. Integration of clinical history, biochemical analysis, urinalysis, and renal ultrasonography facilitates accurate diagnosis and monitoring of disease progression. Continued advances in biomarker research, nephroprotective therapies, and extracorporeal renal support are expected to improve clinical outcomes and reduce mortality associated with this unique form of feline nephrotoxicity.

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