



Acute uremia in cats



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■ Introduction

Acute renal failure may be defined as an abrupt reduction in renal function resulting in accumulation of nitrogenous waste products and dysregulation of water, electrolyte, and acid base balance. "Azotemia" is the accumulation of waste products in the blood, while "uremia" is the polysystemic clinical manifestation of renal compromise.

■ Pathophysiology of acute uremia

Acute renal failure has traditionally been classified as Pre-renal, intrinsic renal, and/or Post-renal in origin. Careful scrutiny of history, physical examination, laboratory findings and imaging studies of cats presenting with signs of an acute uremic crisis usually provides adequate information to identify

Pre-renal, renal and Post-renal components of uremia. Although these categories help establish cause and predict prognosis, they share many pathophysiologic features and are not mutually exclusive. Pre-renal azotemia develops as an adaptive response to any cause of reduced renal perfusion (e.g., hypovolemia, inadequate cardiac output, marked vasodilatation). Initially, nephrons remain intact allowing a rapid return to function once perfusion has been restored. If the patient remains uremic after Pre-renal causes of azotemia are corrected, they should be rapidly and thoroughly evaluated for intrinsic renal and Post-renal causes of azotemia.

Acute intrinsic renal failure occurs when cytotoxic insults to the nephron impair both their structure and function. Acute intrinsic renal failure is commonly caused by nephrotoxins or an ischemic event; other etiologies include infection, prolonged urine outflow obstruction, and severe non-renal systemic disease (e.g., pancreatitis, neoplasia). A recent retrospective study of 32 cats with acute intrinsic renal failure identified nephrotoxins, particularly lilies and non-steroidal anti-inflammatory drugs, as the most common etiologies of intrinsic renal failure in an urban population of cats (1).

Acute intrinsic renal failure may be divided into four sequential stages; 1) initiation, 2) extension, 3) maintenance, and 4) recovery (2). Clinically, transition from one stage to the next may not be clearly evident and not all stages need to be present in an individual patient. Initiation is the period during which the kidneys are exposed to the damaging agent or event. Initiation may last hours to days and is often clinically silent; however, therapeutic intervention at this phase may reduce the severity of renal damage and enhance the likelihood of recovery.

HOW I TREAT...

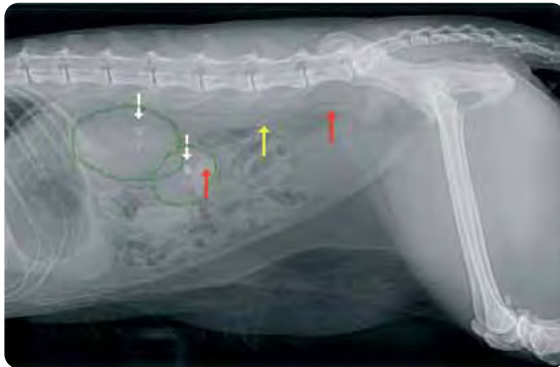


Figure 1. Lateral abdominal radiograph typical of a cat presenting for severe uremia secondary to acute ureteral obstruction. Significant renal asymmetry (Rt > Lt) suggests sequential ureteral obstructions (*i.e.* “big kidney–little kidney syndrome”). Note the presence of bilateral nephroliths (white arrows) and ureteroliths in the right (yellow arrows) and left (red arrows) ureters.

Initiation is followed immediately by extension, during which cytokine-mediated inflammation and alterations in renal perfusion engender tubular epithelial and vascular endothelial injury, culminating in cellular death. During this stage glomerular filtration rate (GFR) declines, urine concentrating ability is lost, and oligoanuria may develop.

Maintenance, the third stage, represents the period of established renal parenchymal damage, and is characterized by persistent reduction of GFR, and tubular dysfunction with variable urine production. Polysystemic signs of uremia become apparent during this phase, alerting owners to seek veterinary care. Unfortunately, significant renal damage may have already occurred thus limiting management to supportive and symptomatic therapies.

The fourth stage, recovery, represents tubular epithelial regeneration, and, if it occurs, may last for days to months. Recovery is characterized by increasing GFR, improved urine quality, and amelioration of the polysystemic consequences of renal dysfunction. These changes may occur gradually or quite precipitously.

Post-renal azotemia results from obstruction of urine flow after it has left the nephron, or leakage of urine from the urinary tract within the body. Classic clinical signs and physical findings of urethral obstruction facilitate rapid diagnosis and relief of the obstruction. Likewise, urinary tract rents may be readily identified using a combination

of physical examination findings and targeted imaging. Obstruction of the upper urinary tract presents more of a diagnostic challenge.

■ Feline ureteral obstruction: an emerging syndrome

Feline ureteral obstruction is currently the leading cause of severe acute uremia in cats (3). Since 1996 approximately half the cats with severe acute uremia hemodialyzed at UC Davis and the UCVMC-San Diego had acute ureteral obstruction. Calcium oxalate stones most often cause ureteral obstruction, although other causes, including concretions of blood and plugs of cellular material or inflammatory debris have also been reported (4).

Unilateral ureteral obstruction rarely causes clinical signs if the contralateral kidney functions well. Perceptive owners may notice flank licking or anti-social behavior, presumably a manifestation of pain, but cats are rarely presented at this stage. Sustained obstruction leads to fibrosis and atrophy of the corresponding kidney and compensatory hypertrophy of the contralateral kidney. The disease remains clinically silent until obstruction of the contralateral ureter occurs. This process explains the classic “big kidney–little kidney” scenario typical of many cats diagnosed with acute ureteral obstruction. Alternatively, some cats have bilaterally small kidneys suggesting ureteral obstruction as an acute exacerbation of chronic kidney disease (CKD). Ureteral obstruction with symmetrical kidneys suggests simultaneous bilateral obstruction (**Figure 1**).

All cats with severe acute uremia should be evaluated for potential ureteral obstruction since return of renal function is inversely related to duration of obstruction. In dogs, many studies have demonstrated complete return of function is possible if the obstruction is relieved within a few days, while less than 50% of the original renal function will return if the obstruction persists for greater than 2 weeks (5).

■ Differentiating acute from chronic disease

The differentiation of acute uremia from CKD has both prognostic and therapeutic relevance. Inherent in the diagnosis of acute kidney injury is the potential for complete functional recovery. Similarly, expedient treatment of factors causing

ACUTE UREMIA IN CATS

acute decompensation of CKD (e.g., pyelonephritis, hypovolemia) may permit reversion to pre-crisis levels of function. In contrast, patients with end-stage CKD lack substantial recovery potential. Meticulous examination of medical history, physical examination, past and current laboratory data and imaging studies will usually enable differentiation.

■ Management of acute uremia

Initial management of cats presenting with acute uremia should focus on reversing the underlying cause(s) (Pre-renal, Post-renal) of the uremia and to identifying and correcting fluid, electrolyte and acid-base imbalances. In addition to specific therapy, should an underlying cause be identified, aggressive supportive and symptomatic care will optimize the chance for recovery of renal function.

■ Fluid balance

Critical to initial management are the establishment and maintenance of euvoemia. Many patients are significantly dehydrated at presentation and rapid restoration of extracellular volume and renal perfusion corrects Pre-renal azotemia and helps prevent further ischemic renal damage. Intravenous (IV) crystalloid rates are calculated to correct extracellular fluid deficits. The calculated deficit should be replaced over 4-6 hours. Maintenance fluid requirements and ongoing losses are added to the deficit calculation to complete the fluid prescription. This is often a significant volume of fluids and patients should be continuously monitored to ensure cardiovascular stability. Normal (0.9%) saline is the fluid of choice for volume deficit replacement. Hypernatremic patients may need lower sodium fluids, while those with hypovolemia, hypotension or blood loss may need colloids or blood products.

The rate and volume of fluid that can safely be given depends on deficit and urine output (e.g., oligoanuria, polyuria). Response to fluids during the rapid rehydration phase must be monitored carefully; oliguria or anuria after volume replacement predisposes to hypervolemia with continued fluid therapy. Body weight must be reliably measured at least twice per day. Rapid gain or loss of 1 kg represents a corresponding gain or loss of one liter of fluid. The quantity of fluid given is adjusted so body weight stays stable. All sources of fluid intake (prescribed fluids, medications, feedings, etc.) and

output (surgical drains, diarrhea, insensible losses, etc.) should be included when considering overall fluid balance. Overhydration is one of the most common, life-threatening, complications encountered in patients with ARF (**Figure 2**).

■ Urine production

Urine production varies significantly between patients with acute uremia, and may vary within a given patient. Normal urine production for a euvoemic, normotensive cat is 1-2 mL/kg/hr. Euvoemia and a mean arterial blood pressure >60 mmHg are essential prerequisites to correctly interpreting urine production. Urine production of < 0.5 mL/kg/hr represents oliguria, and should prompt vigilant patient monitoring. Not all ARF patients develop pathologic oliguria, but it is important to note that normal or increased urine production does not imply normal renal function.

If a patient becomes hypervolemic, *all* parenteral fluid administration must cease and diuretics may be needed. Loop diuretics, most commonly furosemide, diminish active transport and energy requirements in the thick ascending limb of the nephron, and are the most appropriate diuretics for hypervolemia. Although furosemide may increase urine output, human medical trials show no increase in renal recovery or decrease in mortality with its use (6). Regardless of the influence on final outcomes, conversion from oliguria to nonoliguria is important as it greatly facilitates the management

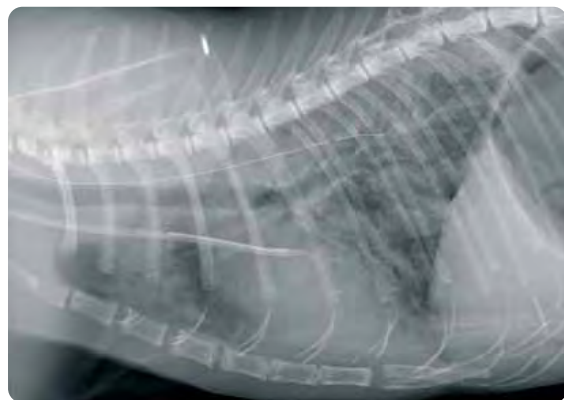


Figure 2. Lateral thoracic radiograph of an oligoanuric cat presented for hemodialysis, demonstrating pulmonary edema secondary to overzealous parenteral fluid administration. In addition to the patchy alveolar lung pattern, note the distention of the caudal *vena cava*; both are typical of overhydration. Note the presence of a dual-lumen jugular catheter for hemodialysis and an esophageal feeding tube.

HOW I TREAT...

of fluid and electrolyte imbalances. This is particularly true in cases where dialysis is not readily available.

If oliguria persists after rehydration, administration of mannitol may promote osmotic diuresis. An initial mannitol bolus of 0.5-1.0 g/kg IV is given over 10 to 20 minutes. If significant diuresis occurs within 60 minutes, the bolus may be repeated every 8 hours. Alternatively, a constant rate infusion of 1-2 mg/kg/min of mannitol may be given for 12-36 hours to sustain the effect. Mannitol increases renal blood flow, decreases tubular cell swelling, increases tubular flow, and helps prevent tubular obstruction and collapse. Mannitol is also a weak vasodilator and a free radical scavenger. Osmotic agents are contraindicated with overhydration, as increasing intravascular volume may precipitate pulmonary edema.

In cats, dopamine infusion, at tolerable doses, has not been shown to increase urine output; probably because cats have few renal dopamine receptors (7). Dopamine is NOT currently a recommended therapy for humans or cats with ARF and its use in dogs is controversial.

Preliminary studies of the selective DA1 agonist, fenoldopam, have shown promising results in humans. There has only been one published study in feline medicine that demonstrated a delayed increase in urine output when administered as a CRI to a group of healthy cats (8).

■ Hypertension

Cats in uremic crisis are frequently hypertensive, which may exacerbate renal injury (9). Careful monitoring of blood pressure and physical examination during fluid resuscitation is essential to prevent hypertension secondary to volume overload. Cats with consistent systolic pressures above 180 mmHg, or those with a systolic blood pressure >160 mmHg and evidence of end organ damage (e.g., retinal arterial tortuosity, hemorrhage or detachment; stroke, or seizure; left ventricular hypertrophy) should be treated for hypertension.

Because of its efficacy, lack of side effects, and once-daily oral administration, the calcium-channel blocker amlodipine currently is the antihypertensive drug of choice in cats (10). Initially 0.625 mg/cat

is given, and this dose is increased as needed to achieve a systolic pressure below 170 mmHg. If blood pressure cannot be controlled with amlodipine, the addition of an angiotensin converting enzyme inhibitor (ACEi) and/or alpha-1 antagonist should be considered.

■ Metabolic complications

Acid-base imbalances

Metabolic acidosis is a common sequela of acute uremia and occurs secondary to reduced renal acid excretion and decreased bicarbonate generation (11). Mild metabolic acidosis may resolve with volume replacement and the onset of diuresis. However, more aggressive correction of metabolic acidosis may be indicated if the acidosis is very severe or if hyperkalemia is also present.

Appropriate treatment of severe acidosis ([serum bicarbonate] <16 mmol/L) is based on the bicarbonate concentration in serum or on venous blood gas evaluation. Sodium bicarbonate is given IV to attain target bicarbonate concentrations (>20 mmol/L) or until sodium overload or hypocalcemia precludes further administration. The bicarbonate deficit in the extracellular fluid may be estimated thus:

$$\text{- mEq HCO}_3 \text{ required} = (\text{Body wt in kg}) \times 0.3 \times (\text{base deficit or } (20 - \text{TCO}_2))$$

To minimize iatrogenic complications, the immediate goal is not to restore acid-base balance, but rather to ameliorate the adverse cardiovascular effects of acidosis. Thus, 1/2 the calculated dose is given over 30 minutes, then the remainder may be given with IV fluids over the next 2-4 hours. Serum or blood gas TCO₂, and electrolytes are reassessed after initial replacement, to assess treatment efficacy and determine need for additional replacement therapy (12).

Hyperkalemia

Hyperkalemia is the most serious metabolic derangement associated with intrinsic renal or Post-renal causes of acute uremia. Hyperkalemia may be aggravated by inappropriate use of potassium containing fluids and/or use of drugs such as ACEi. Serum potassium concentration (serum [K⁺]) varies substantially in acutely uremic patients, and life-threatening cardiac arrhythmias may develop with serum [K⁺] >7 mEq/L. Hypocalcemia, acidosis and certain medications potentiate the

ACUTE UREMIA IN CATS

Table 1.
Therapeutic options for the management of hyperkalemia *

Intervention	Cautions
Clinical	
Correct dehydration with potassium free (0.9% NaCl) fluids	Monitor for hypervolemia and hyponatremia
Encourage diuresis	
Minimize potassium intake (eg. eliminate parenteral sources of potassium, minimize oral intake)	
Discontinue medications that promote hyperkalemia (eg. angiotensin-converting enzyme inhibitors, potassium sparing diuretics)	
Pharmacologic	
Loop diuretics Furosemide 2-4 mg/kg	Monitor blood pressure, acid-base, and hydration status. Only effective if non-oliguric
Sodium bicarbonate Sufficient to correct existing bicarbonate deficit If bicarbonate status unknown, 1-2 mEq/kg IV	Ensure adequate respiratory function Monitor for alkalosis and hyponatremia
Dextrose ± Insulin 1-2 mL/kg of 50% dextrose (diluted to 25%) IV OR Regular insulin 0.1-0.2 U/kg IV bolus followed by 1-2 g dextrose/unit insulin OR 0.5-1.0 U/kg CRI with 2 g dextrose per unit of insulin administered	Monitor carefully for hypoglycemia
Calcium gluconate 0.5-1.0 mL/kg of 10% calcium gluconate IV over 10-15 min.	May cause bradycardia, cardiac arrhythmias Administer with constant ECG monitoring
Refractory hyperkalemia	
Dialytic management	

* Therapy must be tailored to the individual patient. See text for details.

electromechanical effects of hyperkalemia, and the ECG displays the summation of these effects. Initial changes in the pattern of the ECG include peaking of the T waves, followed by QT interval shortening and P wave flattening. As serum $[K^+]$ increases, the P wave is attenuated, the QRS complex widens, and the QT interval becomes prolonged. The eventual development of a sine wave pattern (“idioventricular rhythm”) heralds imminent cardiac arrest (11).

Moderate hyperkalemia (serum $[K^+] < 7.0$ mEq/L) will often resolve with fluid deficit correction using normal saline. Here, the reduction in serum $[K^+]$ is due to hemodilution and increased excretion from improved renal blood flow. Furosemide may also be useful in promoting kaliuresis.

If volume replacement and diuresis does not sufficiently mitigate hyperkalemic cardiotoxicity, other therapies may be needed to temporarily reduce effective serum $[K^+]$ until renal function improves or renal replacement therapy is initiated. The interventions outlined in **Table 1** may prevent or

reverse hyperkalemic cardiotoxicity by decreasing serum $[K^+]$ or, in the case of calcium gluconate, by stabilizing cardiac cell membranes.

If sodium bicarbonate is contraindicated or ineffective, hypertonic dextrose may be given alone or in combination with regular insulin. Glucose stimulates insulin release and promotes cellular potassium uptake. If insulin is given, blood glucose must be closely monitored to avoid iatrogenic hypoglycemia (12).

Calcium gluconate does not alter serum $[K^+]$, but mitigates cardiotoxicity by permitting cardiac cell membrane depolarization in the face of severe hyperkalemia. The recommended initial dosage is 0.5 to 1.0 mL/kg IV of a 10% solution over 10 to 15 minutes, to reverse life-threatening ECG abnormalities. Effects on the ECG are rapid in onset but short-lived, lasting approximately 25 minutes. Calcium infusion is a bridging measure only, enabling prompt application of other more durable therapies (11).

HOW I TREAT...

Hypokalemia

Hypokalemia is commonly associated with polyuric renal failure. Administration of potassium-free fluids (saline) or diuretics (furosemide, mannitol), inadequate dietary potassium intake, vomiting, and diarrhea may all contribute to the development of hypokalemia. Hypokalemia may alter systemic hemodynamics and decrease GFR, causing further renal impairment. Potassium supplementation of crystalloids given to ARF patients is based on serial evaluation of serum $[K^+]$. The rate of IV potassium administration should not exceed 0.5 mEq/kg/hour. Once hypokalemia is corrected, appropriate oral or IV potassium therapy maintains normokalemia.

Sodium imbalance

Acute renal failure deranges normal water and sodium handling. Patients with impaired excretory function require more scrupulous monitoring of administered fluid volume and composition; both hypo- and hypernatremia are common in these patients, and are usually iatrogenic. Oliguric patients are often hyponatremic due to limited renal free water clearance. Hypernatremia may result from hypotonic fluid losses gastrointestinal (GI) loss, osmotic diuresis, insensible losses), administration of sodium-rich fluids (0.9% saline, sodium bicarbonate, lactated Ringer's), and inadequate free water intake. Frequent monitoring of fluid balance and serum sodium concentration, and appropriate adjustment of fluid composition will minimize the occurrence of this complication (11).

Hyperphosphatemia

Marked hyperphosphatemia is a hallmark of acute uremia, and may exacerbate hypocalcemia, promote soft tissue calcium and phosphorous deposition, and further impair renal function. The mainstay of managing hyperphosphatemia is dietary phosphorous reduction in conjunction with administration of enteral phosphate binders (e.g., aluminum hydroxide).

Gastrointestinal manifestations of uremia

Nausea and vomiting are common clinical manifestations of acute uremia that must be controlled to facilitate caloric intake and improve patient comfort. Uremic vomiting is mediated centrally via the effects of uremic toxins on the chemoreceptor trigger zone (CRTZ) in the brain,

and peripherally via GI irritation. Derangements in GI motility and the presence of gut edema (seen with overhydration) may also contribute. Although vomiting and nausea are usually controlled with the use of a single drug, a second antiemetic may be added in cases of protracted vomiting.

Dopaminergic antagonists (e.g. metoclopramide) are commonly used as a first line therapy in the management of uremic vomiting. Metoclopramide also has a promotility affect in addition to its centrally acting anti-emetic effects. The α_2 adrenergic antagonists (e.g. prochlorperazine) are effective anti-emetic agents in cats but have been associated with significant hypotension and/or tranquilization. This class of anti-emetics should only be used in volume replete, normotensive patients with careful blood pressure monitoring. The 5-HT₃ antagonists (ondansetron and dolasetron) appear to be very effective in controlling uremic vomiting and have not been associated with significant adverse effects (11).

Although not yet approved for use in cats, maropitant has recently become available to manage vomiting in dogs. Maropitant is a neurokinin (NK₁) receptor antagonist inhibits vomiting via both peripheral and centrally mediated effects. Preliminary, extra-label use of this drug in cats seems promising, but requires further safety and efficacy evaluation.

Acute gastritis and enteritis may be managed with H₂-receptor antagonists such as famotidine. Since these drugs undergo renal elimination, the dose is reduced in the face of severe renal failure. As an alternative to H₂ receptor antagonists, proton pump inhibitors such as omeprazole may be used to decrease gastric acid secretion, minimizing gastric mucosal irritation.

■ Anemia

Normochromic-normocytic anemia often develops during the management of acute uremia. Humans with ARF develop anemia in the face of normal serum iron levels and normal or hypercellular bone marrow due to the combined effects of decreased endogenous erythropoietin production and uremic toxin-induced red blood cell fragility. Blood loss from gastrointestinal ulcerations,

ACUTE UREMIA IN CATS

potentiated by uremic platelet dysfunction, may also contribute to anemia. Because anemia can produce significant complications in critically ill patients, hemoglobin levels should be maintained above 10 g/dL.

Supplementation with subcutaneous or intravenous recombinant human erythropoietin (rHu EPO) produces an initial erythroid response in most cats. Used chronically, pure red cell aplasia due to anti-rHuEPO antibodies develops in 20-70% of patients precluding further administration (13). Darbepoetin alfa is a novel erythropoietic peptide shown to be as effective as rHuEPO for anemia management in humans with CKD (14). Compared with rHuEPO, darbepoetin has a longer half-life and greater potency, enabling clinical efficacy with less frequent administration. Although not yet proven by clinical trial, anecdotal reports suggest that darbepoetin has similar efficacy and safety to erythropoietin, with the significant benefit of decreased incidence of antibody production.

■ Pain management

Analgesia is an important component of therapy, often overlooked in cats due to their reticent nature. Cats with ureteral obstruction usually have mild to marked mid-cranial abdominal pain, from interstitial edema and/or ureteral spasm; cats with intrinsic ARF often have pain from renal inflammation and/or swelling. In addition, severely uremic cats often have oral and gastrointestinal ulceration. Discomfort from oral erosions and ulcerations may be eased with topical lidocaine-containing agents, and oral rinses with 0.2% chlorhexidine may reduce infection and speed healing. Buprenorphine or butorphanol at standard dosages provide reliable and effective initial analgesia.

■ Pharmacologic considerations

Renal insufficiency can markedly alter the pharmacokinetics of drugs; including oral bio-availability, volume of distribution, protein binding, and most importantly rates of metabolism and excretion (*i.e.*, clearance). To minimize toxicity and maximize benefit, modification of the dosage or administration frequency is often necessary. Guides for dosage adjustment in humans are available, and many drug inserts include dosing regimens for

renally impaired patients. Although drug metabolism may differ between cats and humans, these references may serve as guides.

■ Nutritional management of acute renal failure

Patients with acute uremia are often profoundly catabolic. Additionally, the metabolic acidosis of ARF increases protein catabolism, thereby exacerbating azotemia, hyperkalemia, hyperphosphatemia and loss of lean body mass. Metabolic causes of anorexia and vomiting must be managed aggressively and oral intake of calories should be encouraged. As cats are notoriously finicky eaters, they should be offered diets unrelated to the preferred long term diet when hospitalized (*i.e.*, don't force-feed a renal diet in-hospital). This helps prevent development of food aversion to the preferred therapeutic diet.

Although many hospitalized cats refuse to eat, proactive intervention with enteral feeding tubes combats catabolism and helps prevent loss of lean body mass. Once vomiting is controlled, nasoesophageal feeding tubes provide short-term nutritional and fluid support. Most patients tolerate use of these tubes, through which commercially available liquid feline renal diets may be fed. An esophagostomy tube (E-tube) or percutaneous gastrostomy tube (PEG tube) should be considered if nutritional support is anticipated for more than a few days. At our hospital, E-tubes are routinely placed in uremic cats for long-term provision of nutrition, hydration, and/or medications. Esophagostomy tubes are well tolerated and permit feeding of a blenderized, prescription renal diet. When protracted vomiting or a moribund state precludes enteral feeding, parenteral nutrition may temporarily provide daily caloric requirements. Administration of parenteral nutrition to oliguric animals requires diligent monitoring to avoid fluid and electrolyte imbalances.

■ Indications for dialysis

Hemodialysis or peritoneal dialysis are often the only options for acutely uremic patients unresponsive to appropriate, aggressive medical management. Indications for initiating dialysis include severe hyperkalemia, volume overload refractory to fluid restriction and diuretics, intract-

ACUTE UREMIA IN CATS

able uremia and (especially for hemodialysis) acute toxicities and drug overdoses. The metabolic stability provided by dialysis provides time to determine the cause of renal dysfunction, giving clients better prognostic data. Ideally, dialysis creates a window of stability long enough to enable renal recovery (16). In the earlier stages of progressive azotemia, hemodialysis may also be initiated proactively to forestall or preclude development of uremia. This approach improves quality of the life and owner satisfaction, and facilitates overall case management.

■ Prognosis and outcome

Prognosis for cats with acute uremia depends on underlying cause, extent of renal injury, concomitant disease or organ failure, age, and response to therapy. An overall mortality rate of 47% was recently reported for a group of 32 cats with acute, intrinsic renal failure (1). In this study, approximately half of the cats that survived the uremic episode remained persistently azotemic. These findings parallel those reported for dogs and humans. A retrospective study from 1997 reported that 56% of dogs diagnosed with ARF and receiving non-dialytic management at a university hospital, were euthanized or died before discharge (17). Of the surviving dogs, over half remained persist-

ently azotemic. Three studies of cats presented for medical, and in most cases hemodialytic, management of acute uremia, have shown no association between the magnitude of azotemia at presentation and the outcome (1,3,16). Similar findings were observed in a study of dogs with hospital acquired acute renal failure (18). However, in another study of 99 dogs with ARF, a marked elevation in serum creatinine at presentation was associated with a decreased survival (17).

Cats with acute uremia secondary to acute ureteral obstruction fare better than those with acute intrinsic renal failure; in a report of 50 cats presented for dialytic management of acute ureteral obstruction 70% survived. Of the surviving cats, 71% were azotemic at the time of discharge (19). It is reasonable to hypothesize that an increased percentage of cats surviving an acute ureteral obstruction would remain azotemic due to pre-existing renal disease or the potential of a persistent partial ureteral obstruction.

Overall, the long-term prognosis for cats surviving episodes of acute uremia is fair to good depending on the underlying etiology. Early diagnosis and appropriate intervention improve survival and minimize the potential of persistent renal injury.

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