Management of Urinary Tract Emergencies in Small Animals

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INTRODUCTION

Emergencies involving the urinary tract are commonly encountered in small animal practice, and several of these have the potential to become life threatening if not addressed rapidly. This article focuses on some of the most commonly seen urinary tract emergencies in dogs and cats, with emphasis on basic pathophysiology, diagnosis, and emergency management of these cases. These emergencies can be divided anatomically into conditions that affect:

- The upper urinary tract: kidneys, renal pelvis, ureters
- The lower urinary tract: urinary bladder, urethra

ACUTE KIDNEY INJURY

Acute kidney injury (AKI) is characterized by an abrupt, sustained decrease in renal function and loss of the kidneys’ ability to excrete wastes, regulate acid-base and electrolyte balance, and concentrate urine.1 AKI was previously referred to as acute renal failure (ARF). This recent change in nomenclature reflects a more accurate

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understanding of the histopathologic changes that occur with an acute insult to the kidneys and encompasses a spectrum of alterations in renal function, ranging from mild to severe (Fig. 1). Human studies report incidence rates of AKI of 2% to 7% in all-hospital patients, and nearly 15% in critically ill patients with mortalities near 50%.\(^1\,^2\) Veterinary studies have reported mortalities between 23.8% and 78.3%\(^3\)\(^–\)\(^5\) in dogs and approximately 47% in cats.\(^6\)

Acute onset of azotemia can be prerenal, renal, or postrenal in origin. AKI refers to a complex disorder that comprises multiple causative factors and occurs in a variety of settings with a range of clinical manifestations that range from a minimal but sustained increase in serum creatinine to anuric renal failure. Prerenal azotemia and other fully reversible causes of acute renal insufficiency are specifically excluded from the spectrum of AKI. Prerenal azotemia usually results from decreased renal perfusion, glomerular filtration rate (GFR), and renal blood flow. This condition can be caused by dehydration or systemic hypotension secondary to a decrease in effective circulating volume as occurs in hypovolemia or vasodilatory shock. Postrenal azotemia occurs when there is an obstruction to urine outflow as is seen with renal, ureteral, or urethral obstructions, or urine leakage caused by loss of integrity of some portion of the urinary tract. Intrinsic renal failure in dogs and cats can have a wide variety of causes and occurs when there is damage to the renal parenchyma. These causes can be classified as:

1. Toxic:
   a. Ethylene glycol (EG)
   b. Nonsteroidal antiinflammatory drugs (NSAIDs)
   c. Aminoglycoside antibiotics
   d. Lilies (cats)
   e. Grapes and raisins
   f. Heavy metals
   g. Amphotericin B
   h. Phosphate enemas
   i. Polymyxin B
   j. Sulfonamides
   k. Intravenous (IV) contrast agents
   l. Tetracyclines
   m. Mushrooms

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**Fig. 1.** Pathophysiology of intrinsic acute kidney injury (AKI). GFR, glomerular filtration rate.
2. Infectious:
   a. Leptospirosis
   b. Pyelonephritis
   c. Rocky Mountain spotted fever
   d. Glomerulonephritis
   e. Borreliosis

3. Others:
   a. Cardiovascular shock
   b. Systemic hypotension
   c. Burns/heatstroke
   d. Postcardiac arrest syndrome
   e. Thromboembolic disease
   f. Sepsis
   g. Anaphylaxis
   h. Prolonged general anesthesia
   i. Pigment nephropathy
   j. Transfusion reactions
   k. Snakebites
   l. Traumatic injury

PATHOPHYSIOLOGY OF AKI

AKI in humans is staged using the risk, injury, failure, loss, end-stage renal disease (RIFLE) acronym. Serum creatinine levels and urine output (UOP) are the two most important markers used to stage AKI in people (Table 1). Another staging system, the Acute Kidney Injury Network (AKIN) criteria has also been recently used in human medicine (Table 2). A recent study from 2011 evaluated a new staging system for AKI in veterinary medicine called the Veterinary Acute Kidney Injury (VAKI) scheme; this also uses increases in serum creatinine to stage patients. The VAKI system stages dogs with AKI on a scale from 0 to 3, with 0 being the least severe and 3 being the most severe. Table 3 provides more information on the VAKI classification system.

<table>
<thead>
<tr>
<th>Table 1</th>
<th>RIFLE staging system</th>
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<tbody>
<tr>
<td></td>
<td>Urine Output</td>
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<tr>
<td>Risk</td>
<td>Decrease in GFR ≥25%; &lt;0.5 mL/kg/h for ≥6 h</td>
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<tr>
<td>Injury</td>
<td>Decrease in GFR ≥50%; &lt;0.5 mL/kg/h for ≥12 h</td>
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<tr>
<td>Failure</td>
<td>Decrease in GFR ≥75%; &lt;0.3 mL/kg/h for ≥24 h or anuria ≥12 h</td>
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<tr>
<td>Loss</td>
<td>Persistent acute renal failure: complete loss of kidney function for &gt;4 wk</td>
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<tr>
<td>End stage</td>
<td>Complete loss of kidney function for &gt;3 mo</td>
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Ischemic injury is one of the most common causes of AKI, particularly in critically ill patients that are hospitalized. This condition may be caused by a reduction in effective renal perfusion caused by reduced intravascular volume (eg, hemorrhage, gastrointestinal [GI] or renal losses, third spacing caused by capillary leak), reduced cardiac output (eg, cardiogenic shock, congestive heart failure [CHF], pulmonary hypertension, pulmonary thromboembolism [PTE], pericardial disease), systemic vasodilation (eg, anaphylaxis, sepsis), or renal vasoconstriction (eg, contrast nephropathy, vasopressor medications, NSAIDs).

**DIAGNOSIS OF AKI**

Diagnosis of AKI is usually based on history, in conjunction with physical examination (PE) findings and documentation of azotemia (which may be accompanied by oliguria or anuria in advanced cases of the disease). Various electrolyte and acid-base derangements can be seen with AKI including hyperkalemic or hypokalemia, hyperphosphatemia, and metabolic acidosis. Acutely uremic animals can also present with signs of systemic illness including lethargy, inappetence, vomiting, diarrhea, and halitosis. Uremic ulcers may be seen in these patients. Neurologic signs are also sometimes observed in these patients and may be attributed to uremic encephalopathy.

AKI can be present even if the serum creatinine is within the normal reference range, especially in hospitalized patients. A 2005 study in humans showed that even a mildly increased creatinine concentration of 0.3 mg/dL from baseline increased the risk of

| Stage 2 | <0.5 mL/kg/h for ≥12 h | >200%–299% increase from baseline serum creatinine |
| Stage 3 | <0.3 mL/kg/h for ≥24 h or anuria ≥12 h | ≥300% increase from baseline serum creatinine or absolute serum creatinine ≥354 μmol/L (4.0 mg/dL) with an acute increase of ≥44 μmol/L (0.5 mg/dL) |


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**Table 2**

<table>
<thead>
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<th>AKIN staging system</th>
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<tbody>
<tr>
<td>Urine Output</td>
</tr>
<tr>
<td>Stage 1</td>
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<tr>
<td>Stage 2</td>
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<tr>
<td>Stage 3</td>
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</table>

death by as much as 70%. This is important to bear in mind because it may help identify patients with clinically significant kidney injury that may not otherwise be identified. For example, a patient whose creatinine increases from 1.5 mg/dL to 1.8 mg/dL would be in the category of stage I in the VAKI staging system. Such small increases in serum creatinine, although clinically significant, can be difficult to detect consistently because of the possibility of intermachine and intersample variability. Therefore, ideally, the same machine (one that has high precision) should be consistently used to monitor kidney values in an animal suspected of having AKI to allow detection of even the smallest changes.

GENERAL MANAGEMENT OF AKI

- The management of AKI depends largely on the underlying cause. However, the ultimate goal in all cases is to optimize hemodynamic status, restore adequate perfusion to the kidneys, and limit further injury to the renal tubules by reversal of the underlying cause.
- Aggressive IV fluid therapy to promote diuresis and reverse azotemia is the hallmark of treatment of AKI. There has been extensive research in human medicine with regard to the best type of fluid to use in cases of AKI. One of the largest studies, the Saline versus Albumin Fluid Evaluation (SAFE) study,11 showed that albumin transfusion was safe, but not any more effective than isotonic saline in preventing death or the need for dialysis in patients in the intensive care unit (ICU). Other studies have corroborated the use of crystalloids by showing no difference between crystalloids and colloids in treating AKI. Synthetic colloids have been linked with causing or worsening AKI in several human studies in critically ill patients.12,13
- Because AKI is characterized by a spectrum of fluid responsiveness, close monitoring of patients is imperative to help guide therapy and detect deteriorations in renal function. Early clinical recognition of the presence of urinary casts and glucosuria may be a marker for tubular injury.
- Another upcoming area of research involves measurement of various biomarkers that are released into the blood or urine by the injured kidney at an early stage of damage. This indicator of early disease may help initiation of therapies that can stem progression or repair damage. Biomarkers ideally should be able to differentiate incipient acute tubular necrosis from other forms of acute renal dysfunction (eg, volume-responsive AKI; acute glomerular, vascular, and interstitial diseases; obstructive nephropathies), allow monitoring of the effects of treatment, and predict the need for dialysis, long-term kidney outcome, and mortality. Examples of these include interleukin 18, neutrophil gelatinase-associated lipocalin, kidney injury molecule-1, and liver fatty-acid-binding protein.14,15 Occasional increases in the levels of these markers in the urine can occur even before serum creatinine increases.14,15
- Depending on the nature of injury and severity of illness, patients should be monitored closely for urine production. Placement of indwelling urinary catheters is useful in this regard, especially in patients that are tending toward oliguria or are already anuric. However, caution should be exercised before placing catheters in patients with evidence of an active urinary tract infection, in diabetic patients, in immunocompromised patients, or in patients with a coagulopathy that are at risk for bleeding. Ascending nosocomial infections are also a concern, particularly in patients already on systemic antibiotic therapy and even in otherwise healthy patients.16
- Careful assessment of the patient, including PE findings and monitoring tools (eg, central venous pressure, urine output monitoring, weight gain or loss) should be used. In patients showing decreased UOP, the use of diuretics and other therapy may be necessary. Fig. 2 shows guidelines for fluid therapy for patients with AKI, and Table 4 gives a listing of diuretics used with AKI.

MANAGEMENT OF COMMON CONDITIONS CAUSING AKI

**EG Intoxication**

EG is a common cause of toxicity in dogs and cats, and is found in multiple sources (eg, antifreeze, printer cartridges, paint, caulking material). In general, toxicosis is only seen after ingestion of antifreeze that is typically greater than 90% EG and concentrated (compared with other products that contain <1%–2% EG). EG toxicity can be fatal, with mortalities in cats ranging from 96% to 100% and 59% to 70% in dogs.\(^{19-22}\) Cats also have a significantly lower minimum lethal dose compared with dogs (1.4 mL/kg compared with 4.4–6.6 mL/kg in dogs).\(^ {19-21}\)

EG by itself is nontoxic; it is the dangerous metabolites that result in severe AKI. With EG ingestion, absorption is rapid, with peak blood levels occurring about 3 hours after ingestion and clinical signs developing within 20 to 30 minutes. EG is metabolized by the enzyme alcohol dehydrogenase (ADH) into several toxic metabolites that are responsible for clinical signs. The first metabolite, glycoaldehyde, is produced after metabolism by ADH, which is the first rate-limiting step of this reaction. This metabolite is then converted to glycolic acid, which is then rapidly oxidized to glyoxylic acid (the second rate-limiting step). Glycolic acid then accumulates, resulting in metabolic

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**Assess patient’s intravascular volume status:**

- Physical examination findings: Perfusion parameters (e.g., heart rate, CRT, mucous membrane tachiness, pulse quality?), weight gain or loss
- Blood work findings: Elevated lactate, elevated PCV/Ts? Hyperthrenuria?
- Blood pressure: Increased or decreased?
- Imaging: Size of the heart and vena cava on thoracic radiographs?

**Central venous pressures:** High, low, or normal?

- **Hypovolemia**
  - Continue with IV fluid therapy until volume replete and normotensive
  - Target CVP: 2-8 cm H\(_2\)O (dogs), 2-4 cm H\(_2\)O (cats)
  - If UOP still decreased at this stage, consider diuretic therapy. Typical first choices include furosemide or mannitol

- **Normovolemia/Volume overload**
  - Consider starting a diuretic. Typical first choices include furosemide or mannitol
  - If UOP still inadequate and azotemia not improving, consider adding in a second diuretic.

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Fig. 2. Fluid therapy guidelines in AKI. CRT, Capillary Refill Time; CVP, Central Venous Pressure; PCV, Packed Cell Volume; TS, Total Solids.
acidosis. Glyoxylic acid is further metabolized to oxalates, formic acid, serine, glycine, and carbon dioxide. Oxalates, along with glycolic acid, are the main causes of acute tubular necrosis. Oxalates combine with calcium to form calcium oxalate monohydrate crystals within the lumen of the renal tubules, resulting in severe AKI.

Clinical signs from EG toxicity can be caused both by unmetabolized EG and its toxic metabolites. Unmetabolized EG causes signs similar to those seen with ethanol intoxication including GI (eg, nausea, hypersalivation, vomiting), renal (eg, polyuria, polydipsia), and neurologic signs (eg, ataxia and mentation changes ranging from depression to stupor). These signs occur soon after ingestion and last about 12 hours with oliguric renal failure developing within 12 to 24 hours after ingestion in cats, and 36 to 72 hours after ingestion in dogs.

Management

- Emergent treatment of EG ingestion includes decontamination via induction of emesis (if the patient is conscious and able to vomit). However, the rapid absorption of EG limits the effectiveness of this option. Emesis induction is contraindicated if the patient is already showing GI signs (such as vomiting) or neurologic signs (such as mentation changes or seizures), given the risk for causing aspiration of gastric contents. Decontamination using activated charcoal is of little use because EG is not significantly adsorbed by charcoal.
- Treatment of EG intoxication involves inhibiting metabolism of EG into its toxic metabolites and promoting excretion of unchanged EG by promoting diuresis. Treatment with the antidote (eg, either 4-methylpyrazole or ethanol) must be started within 3 hours in cats and 8 to 12 hours in dogs to ensure survival. If renal azotemia has already developed, this treatment is unlikely to be successful.
  - Ethanol has traditionally been used as a competitive substrate that is capable of binding to the enzyme ADH, thereby preventing the enzyme from acting on EG and producing its toxic metabolites. Ethanol can be administered IV as a 20% solution at a dose of 5 mL/cat initially, repeated every 6 hours for 5 treatments, and then every 8 hours for 4 treatments. Risks with using ethanol include possible exacerbation of neurologic signs (eg, drunkenness, obtundation), hypoglycemia (secondary to the ethanol), and worsening of serum hyperosmolality induced by EG.
  - 4-Methylpyrazole (4-MP; also called fomepizole) is another compound that is used as a direct inhibitor of ADH and can be used when hemodialysis is not available or administered while hemodialysis is being set up. 4-MP forms a complex with ADH and blocks the EG binding site, thereby preventing its metabolism. For dogs treated with 4-MP, 1 of 2 dosage regimens may be used: 20 mg/kg of body weight given IV initially, 15 mg/kg 17 hours later, and 5 mg/kg 25 and 36 hours after the initial dose, or 20 mg/kg IV initially, 15 mg/kg 12 and 24 hours later, and 5 mg/kg 36 hours after the initial dose. In cats, it can be administered at a dose of 125 mg/kg IV initially, followed by intermittent doses of 31.25 mg/kg at 12, 24, and 36 hours respectively. A study published in 2010 evaluating the use of 4-MP in EG intoxication in cats showed that 4-MP was safe to use, and, when administered within 3 hours of ingestion, prevented fatal ARF induced by EG. This treatment is usually not combined with hemodialysis because 4-MP is readily dialyzed.
  - Hemodialysis is recommended for cases of ARF induced by EG to remove the circulating EG and its metabolites, as well as to filter out uremic wastes. If resources are available, hemodialysis should be considered even before there is overt evidence of ARF to ensure the best possible prognosis.
<table>
<thead>
<tr>
<th>Drug</th>
<th>Mechanism of Action</th>
<th>Adverse Effects</th>
<th>Dose</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide</td>
<td>Loop diuretic; blockade of the Na-K-2Cl transporter on the luminal side of the thick ascending loop of Henle; inhibits sodium transport, reducing energy requirements of cells in the medullary thick ascending limb the loop of Henle; reduces tubular-glomerular feedback to prevent a decrease in GFR, flushes out intratubular casts, reducing tubular obstruction</td>
<td>Dehydration/volume contraction and prerenal azotemia; electrolyte abnormalities; hypochloremic metabolic alkalosis; ototoxicity at high doses</td>
<td>Wide range: 0.25–4 mg/kg IV bolus in dogs; 0.25–2 mg/kg IV bolus in cats. CRI at 0.1–2 mg/kg/h may be used thereafter, pending patient response</td>
<td>Typically used as a first-line diuretic in oliguric or anuric renal failure to increased UOP and tubular flow</td>
</tr>
<tr>
<td>Mannitol</td>
<td>Osmotic diuretic; acts at the proximal tubule and loop of Henle by extracting water from intracellular compartments; improves tubular flow; free radical scavenger; preserves mitochondrial function by reducing postischemic swelling; improves renal blood flow by inhibiting renin release, expanding intravascular volume, and reducing blood viscosity</td>
<td>Dehydration/volume depletion; hypernatremia; osmotic nephrosis with prolonged use (causes swelling of tubular epithelial cells caused by vacuole formation in the cytoplasm)</td>
<td>0.5–1.0 g/kg slow IV bolus over 20 min followed by CRI at 60–120 mg/kg/h for 24–48 h</td>
<td>Ineffective in anuric renal failure. Risks of volume overload. Cautious use in hyperkalemic animals or those with potential for volume overload or hypernatremia</td>
</tr>
<tr>
<td>Drug</td>
<td>Description</td>
<td>Hypotension</td>
<td>Dosage</td>
<td>Notes</td>
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<tr>
<td>Diltiazem</td>
<td>Calcium channel blocker; reverses renal vasoconstriction by pregglomerular dilation; inhibition of tubuloglomerular feedback–induced pregglomerular vasoconstriction; cytoprotective effect by preventing mitochondrial calcium accumulation; reversal of thromboxane A2–induced renal vasoconstriction</td>
<td>0.1–0.5 mg/kg slow IV, followed by 1–5 μg/kg/min CRI</td>
<td>Can be used in dogs with AKI secondary to leptospirosis infection; monitor ECG and blood pressure during and following administration</td>
<td></td>
</tr>
<tr>
<td>Fenoldopam</td>
<td>Selective DA-1 agonist (no DA-2 or α receptor effects). Produces systemic and renal vasodilation; increases renal blood flow; direct reduction of sodium reabsorption in the proximal tubule and cortical collecting duct</td>
<td>0.8 μg/kg/min IV CRI (based on experimental studies in beagles)</td>
<td>—</td>
<td></td>
</tr>
<tr>
<td>Dopamine</td>
<td>Increases renal blood flow; induces diuresis and natriuresis via action on DA-1 receptors</td>
<td>May paradoxically cause renal vasoconstriction and impair GFR because of stimulation of DA-2 and α receptors</td>
<td>2.5–5 μg/kg/min IV CRI</td>
<td>Not recommended for routine use; no evidence in the literature that suggests a benefit, particularly in cats</td>
</tr>
</tbody>
</table>

*Abbreviation: ECG, electrocardiogram.*
Supportive care, including IV fluid therapy, antiemetic therapy, serial monitoring of blood glucose (particularly if ethanol is used as an antidote), GI support (eg, antacids, gastric acid reducing medications), and symptomatic supportive care are imperative.

**Acute Pyelonephritis**

Pyelonephritis is defined as inflammation of the renal pelvis and kidneys that is caused by an ascending or hematogenously acquired infection of the urinary tract. Pyelonephritis is a serious and life-threatening complication of lower urinary tract infections, and can cause severe systemic illness in animals, with potentially fatal consequences. If not treated appropriately and in a timely manner, irreversible AKI can ensue.

Depending on the severity of illness, animals with pyelonephritis can present with clinical signs of:

- Lethargy
- Inappetence or anorexia
- Vomiting
- Fever
- Renal pain
- Hematuria
- Stranguria
- Halitosis
- Polyuria/polydipsia

Pyelonephritis that progresses to cause urosepsis (defined as systemic inflammatory response to infection arising from the urinary tract) can present with more severe signs including signs of septic shock such as fever, hypotension, and cardiovascular collapse.

Acute pyelonephritis can be diagnosed based on a combination of history, clinical signs and PE findings, clinicopathologic findings, and advanced diagnostics (eg, abdominal ultrasound, pyelocentesis). Laboratory findings may reveal azotemia and other markers of AKI, leukocytosis (with possible left shift and toxic change), hemconcentration secondary to dehydration, and hypoglycemia (secondary to urosepsis, and so forth). Urinalysis typically reveals evidence of infection (eg, increased white blood cell count with or without obvious gross bacteruria). Abdominal imaging, such as an abdominal ultrasound, can reveal evidence of acute nephritis including enlarged kidneys with decreased corticomedullary distinction, pylectasia, and perirenal effusion (**Fig. 3**). Urine samples obtained via cystocentesis or, ideally, via pyelocentesis, should be cultured for confirmation of infection. Occult pyelonephritis may occasionally occur, wherein urine cultures may be negative, but pyelonephritis may still be present.

**Management**

- Animals presenting with signs of severe sepsis or septic shock should be stabilized with aggressive IV fluid therapy as indicated. Isotonic crystalloids are typically preferred for initial fluid resuscitation in these patients. Following adequate volume resuscitation, continued aggressive IV fluid therapy (eg, >2–4 mL/kg/h) should be used to promote diuresis and improve azotemia. However, caution must be exercised in using high fluid rates in animals with preexisting cardiac or pulmonary disease, or those that are oliguric or anuric, to prevent volume overload.
- Once a diagnosis of pyelonephritis is suspected, a urine sample should be collected via cystocentesis and submitted for culture and sensitivity before starting treatment with broad-spectrum antimicrobials.
Empiric antimicrobial therapy is selected based on the patient’s medical history and prior antibiotic use, and clinical suspicion for the type of infection a patient is likely to have. In dogs and cats, infections with *Escherichia coli*, *Staphylococcus* spp, *Proteus* spp, and *Enterococcus* spp are commonly seen, although several other bacterial species have been documented. Pending culture results, the use of Gram staining of urine sediment can be used to help tailor appropriate antibiotic therapy.

Antibiotics that concentrate well in urine and the renal tissues should be selected.
- These antibiotics include most β-lactams and trimethoprim-sulfate.
- Fluoroquinolones are excreted in the urine predominantly in the active form, so these can be used at higher doses (10–15 mg/kg IV or by mouth, every 24 hours) in dogs but should be reserved for animals that have a prior history of more resistant organisms being cultured from their urine. Fluoroquinolones should be used cautiously in cats, because retinopathies have been documented at doses of more than 5 mg/kg/d, particularly in cats with underlying renal insufficiency.
- After initial antimicrobial therapy is initiated, culture and sensitivity results should be evaluated to guide further therapy and alterations as necessary.

Appropriate analgesia is an important part of the management protocol for acute pyelonephritis because pain can contribute to increased morbidity in hospitalized patients and slow recovery times. Opioids are among the most commonly selected analgesics for this purpose, such as pure μ agonists (fentanyl at 2–5 μg/kg/h, methadone at 0.1–0.3 mg/kg IV every 6–8 hours, hydromorphone 0.005–0.1 mg/kg IV every 8 hours) and partial μ agonists (butorphanol at 0.1–0.3 mg/kg IV every 6–8 hours). Butorphanol Continuous Rate Infusions (CRI) can also be used at 0.1 to 0.4 mg/kg/h. Buprenorphine is also widely used, particularly in cats with doses ranging from 0.01 to 0.03 mg/kg IV every 6 to 8 hours. The use of NSAIDs is generally contraindicated in patients with pyelonephritis, especially if they are hemodynamically unstable. Because of the potent renal artery vasoconstriction that can occur with NSAIDs (caused by the blocking of the locally produced prostaglandins), their use may further decrease perfusion to an already diseased kidney, thereby risking worsening renal function.
The prognosis for pyelonephritis varies with the severity of illness, presenting clinical signs and response to fluid and antibiotic therapy. In general, animals that present with severe signs of urosepsis and septic shock have a worse prognosis than animals that are only mildly azotemic. Early recognition and initiation of rational antimicrobial therapy and close monitoring are important factors that can influence outcome with this disease process. Antibiotics are typically continued for up to 2 to 4 weeks after discharge from the hospital. Repeat urine cultures should be checked between 3 and 7 days after completion of antibiotic therapy to ensure that the infection has cleared. Renal values should also be rechecked at this time to ensure that the azotemia is resolving appropriately.

**Ureteral Obstruction**

Ureteral obstruction is uncommon, but is still a significant cause of urinary obstruction and azotemia in dogs and cats. Ureteral calculi and ureteral strictures are among the most common causes implicated in this condition. Based on a 2005 study evaluating 163 cats with ureteral stones, most obstructions were linked to calcium oxalate calculi. In this study, 87% of the cats that had stone analysis performed had exclusively calcium oxalate stones. Other less common causes of ureteral obstruction include iatrogenic ligation (during ovariohysterectomy), neoplastic obstructions, and blood clots.

Animals with ureteral obstruction may show nonspecific clinical signs ranging from mild to severe, depending on whether 1 or both ureters are obstructed. These signs may include anorexia or inappetence, lethargy, vomiting, weight loss, inappropriate urination or thirst, halitosis, oliguria, or anuria. Animals that are bilaterally obstructed can present extremely ill with more advanced clinical signs, along with anuric renal failure.

Diagnosis of ureteral obstruction is usually difficult on PE and typically requires imaging. Plain abdominal radiographs, urinary tract ultrasonography, and contrast radiography (antegrade pyelography) have all been described, with both antegrade pyelography and computed tomography (CT) being extremely sensitive and specific. Ultrasonography has been shown to be effective at diagnosing hydroureter and hydronephrosis secondary to ureteral obstruction, and is more commonly available.

**Management**

- Emergency stabilization with IV fluid therapy and correction of any electrolyte or acid-base disturbances is indicated in animals that present critically ill.
- Medical and surgical options exist for treatment of ureteral stones. Medical management options include attempting diuresis with IV fluids, whereas surgical options include ureterotomy, pyelotomy, ureteral resection, and reimplantation.
- Amitriptylline, a tricyclic antidepressant, has been shown to help facilitate passage of calculi through the urinary tract by causing smooth muscle relaxation at 1 mg/kg by mouth every 24 hours in an experimental study; however, no clinical data exist supporting its use.
- Mannitol, an osmotic diuretic, can also be used to help open the ureters during fluid therapy. A mannitol CRI can be used at 1 mg/kg/min for 24 hours, after a loading bolus of 0.2 to 0.5 g/kg over 20 to 30 minutes.
- Ureteral stenting has recently become more widely available as a less invasive option to treat ureteral obstructions.

**Uroperitoneum**

Uroperitoneum (or uroabdomen) is an emergency condition in which urine excavates into the peritoneal cavity, resulting in life-threatening electrolyte and acid-base...
derangements. Severe dehydration, hypovolemia, hyperkalemia, metabolic acidosis, profound azotemia, and severe chemical peritonitis are all consequences of uroabdomen.

Common causes of uroperitoneum in veterinary medicine include:

1. Rupture of the urinary bladder either caused by trauma or overdistension secondary to feline urethral obstruction (FUO). Iatrogenic rupture secondary to bladder palpation or cystocentesis can also occur.36
2. Rupture or tear of the intrapelvic portion of the urethra secondary to trauma or iatrogenic injury secondary to aggressive catheterization.
3. Ureteral rupture secondary to trauma or ureteral avulsion (along with concurrent damage to the peritoneal lining of the retroperitoneum) can cause urine leakage into the peritoneum.

Clinical signs of uroperitoneum include lethargy, anorexia, vomiting, severe abdominal pain (with secondary aggression), stranguria, hematuria, and possibly abdominal distension with a palpable fluid wave. The diagnosis of uroperitoneum should be based on PE findings and associated clinical signs mentioned earlier, clinicopathologic findings, abdominal radiographs with contrast, focused assessment of sonography in trauma (FAST) ultrasound or abdominal ultrasound, and by diagnostic abdominocentesis. The abdominocentesis can be performed either blindly or using ultrasound guidance to determine whether the free peritoneal fluid is urine. Once abdominal effusion is obtained, microscopic evaluation of the fluid should be assessed for the presence of a possible urinary tract infection, because leakage of infected urine could lead to secondary septic peritonitis. Next, abdominocentesis fluid samples should be compared for abdominal fluid blood urea nitrogen (BUN), creatinine, and potassium levels to concurrently drawn serum creatinine and potassium levels. Higher levels of BUN, creatinine, and potassium in the abdominal fluid are consistent with uroperitoneum. Fluid ratios should also be assessed for diagnostic evaluation. An abdominal fluid creatinine concentration/peripheral blood creatinine concentration ratio of greater than 2:1 is considered to predict uroabdomen in dogs, whereas an abdominal fluid potassium concentration/peripheral blood potassium concentration of greater than 1.4:1 also predicts uroabdomen in dogs.37 In addition, contrast radiographic studies of the urinary tract, such as intravenous pyelography or cystourethrography, can help show loss of integrity of a particular part of the urinary tract and identify sites of leakage for surgical repair or medical management.

Management

- Initial cardiovascular stabilization with IV fluid therapy should be instituted as necessary, because these patients can present critically ill with signs of shock and perfusion abnormalities. Isotonic crystalloids can be used for initial stabilization with bolus doses ranging between 10 and 50 mL/kg, depending on the severity of the cardiovascular compromise.
- Electrocardiogram (ECG) monitoring and treatment of hyperkalemia should be initiated as necessary.
- Urine drainage from the abdomen is the next step after cardiovascular stabilization has been performed. Continuous passive drainage should be established to achieve stabilization and effective diuresis.
  - Placement of a percutaneous transabdominal drainage catheter such as a pigtail catheter or commercially available peritoneal dialysis catheters allows rapid removal of accumulated urine. Catheter placement should be performed by adhering to sterile technique to avoid iatrogenic introduction of infection.
into the peritoneal cavity. The catheter is then attached to a sterile closed
collection drainage system and the amount of fluid collected can be quantified
and monitored (Fig. 4 shows a sample pigtail transabdominal catheter).

- An indwelling catheter should also be placed in the urinary bladder to keep the
  urinary bladder decompressed and reduce the hydrostatic pressure that may
  promote urine leakage into the abdominal cavity.
- Hemodialysis or peritoneal dialysis may be considered for extremely sick and
  uremic patients until more definitive surgical correction can be performed.
- Surgical exploration and correction is the definitive treatment of large defects;
  small tears in the bladder and urethra can potentially heal on their own. Patients
  should not undergo surgery until stable; a delay of 8 to 12 hours is appropriate if
  necessary to stabilize the patient’s electrolytes, pain, and dehydration.

Overall, the prognosis for survival with a uroabdomen depends on the severity
and location of the condition, degree of metabolic derangements caused, and rapidity
of correction of these abnormalities, as well as concurrent medical problems. Various
studies have reported mortalities of between 42.3% and 56.2% in dogs and 38.4% in
cats.37 The prognosis for uroabdomen remains guarded, but, with early diagnosis
and aggressive, rapid management and stabilization, the prognosis can be improved.

**FUO**

FUO is among the most commonly seen urinary tract emergencies in cats, with several
studies reporting between 2% and 13% incidence rates at veterinary teaching hos-
pitals across North America.38 This condition most commonly affects young male
cats that seem to be predisposed because of their long, narrow urethras, although
it can occur in female cats as well. Several risk factors for FUO have been proposed,
including urolithiasis, lower urinary tract infections, environmental stress, and
crystalluria.38–41

There are various causes that have been implicated; however, idiopathic cystitis
progressing to FUO seems to be the most common, with an incidence of more than
50% reported in a 2008 study.42 Urethral mucus plugs are also commonly implicated
as the cause for FUO, although urinary calculi are also sometimes seen. Urethral plugs
are formed when proteinaceous material leaking from an inflamed urinary bladder

![Fig. 4. Pigtail catheter placed for abdominal fluid drainage.](856)
combines with crystals. These plugs can also comprise organic material such as tissue and red blood cells combined with aggregates of crystalline material.

Clinical signs of FUO include vomiting, stranguria, hematuria, pollakiuria, vocalization, excessive grooming or licking of the perineal area, lethargy, and more severe systemic signs (depending on the severity and duration of obstruction). PE findings may include a firm, distended, nonexpressible, and possibly painful urinary bladder. Absence of a palpable bladder in a cat presenting with these clinical signs does not necessarily rule out an FUO, because bladder rupture may have occurred; in this situation, albeit rare, cats present severely ill. Discoloration of the tip of the penis or presence of urethral mucus plugs (at the tip of the penis) may also be found on PE with FUO.

Most cats presenting with FUO are stable; however, some can present with severe cardiovascular compromise and collapse, often secondary to life-threatening electrolyte and acid-base abnormalities. Hyperkalemia, ionized hypocalcemia, and metabolic acidosis are the most commonly seen derangements. One study reported an incidence of 12% for hyperkalemia, and 6% with severe acidemia (pH <7.10).40 Clinical signs associated with systemic electrolyte and acid-base abnormalities include dehydration or hypovolemia, bradycardia, the presence of arrhythmias, hypothermia, and weak pulses.40,43,44

Emergency management

- Initial cardiovascular stabilization:
  - Cardiovascular stabilization is vital in critically ill cats that are obstructed because they often present with acid-base and electrolyte derangements that can be rapidly fatal. These cats are also cardiovascularly severely compromised and can have decreased tissue perfusion secondary to both hypovolemia and cardiac dysfunction from electrolyte derangements (eg, hyperkalemia).
  - Establishing IV access: an intravenous catheter should be placed and blood drawn for an emergency database screen including a venous blood gas, Packed Cell Volume (PCV), Total Solids (TS), BUN, creatinine, glucose, serum sodium, potassium, and ionized calcium values if possible.
  - Animals presenting with signs of hypovolemia or poor tissue perfusion should be stabilized with IV fluids. A bolus of between 10 and 30 mL/kg of a balanced isotonic crystalloid is typically chosen as a starting point, followed by frequent reassessment of the patient.
  - An ECG recording should be obtained as soon as possible, especially in animals that present bradycardic or excessively tachycardic and have irregular or weak pulses. Typical arrhythmias observed in sick, hyperkalemic animals have been inconsistent with those classically observed in experimental hyperkalemia and were described in a 2008 study.45 Some of the expected ECG changes with hyperkalemia are shown in Table 5.
  - Immediate management of arrhythmias secondary to hyperkalemia is needed. Table 6 shows therapeutics for hyperkalemia.
  - Once the patient has been stabilized, therapeutic relief of the urinary obstruction should immediately follow. Table 7 lists various sedation protocols, and Figs. 5–8 show protocols for relief of FUO.

Relief of urinary obstruction

After cardiovascular stability is achieved, the FUO should be relieved.

- Local anesthetic agents may be used to perform epidural blocks in extremely unstable animals to avoid the risk of general anesthesia, and provide relief
from penile and urethral pain. A recent study described the technique for performing a coccygeal epidural block using lidocaine before catheterizing blocked cats.46

- Various types of urinary catheters are available for relief of the obstruction, including the standard Tom Cat, olive-tipped, polyurethane (with or without stylets for insertion), or Slippery Sam catheter.
- Antegrade urethral catheterization:
  - Fluoroscopically-guided percutaneous antegrade urethral catheterization has recently been described in blocked cats where traditional catheterization methods have been unsuccessful.47
  - In this procedure, an IV catheter is placed percutaneously transabdominally into the urinary bladder. Following this, iodinated contrast material is injected and a cystourethrogram performed to assess the integrity of the lower urinary tract.
  - A hydrophilic guidewire is then inserted through the catheter and, using fluoroscopy, guided down into the urethra and advanced in an antegrade fashion, exiting through the urethral tip. Through-and-through guidewire access of the lower urinary tract is thus achieved.
  - Following this, an over-the-wire urinary drainage catheter, or a 5-Fr red rubber catheter can then be passed over the wire into the urethra in a retrograde fashion into the bladder, and the guidewire can be removed through the tip of the urethra.
  - The catheter is then sutured in place routinely, and the IV catheter placed percutaneously into the bladder is removed.
- Postcatheterization care:
  - In cases in which an indwelling catheter is left in place, cats should be monitored in hospital for the next 12 to 48 hours with aggressive IV fluid therapy and analgesia (Fig. 9)
  - Regular monitoring of blood work (if financial limitations exist, treatments such as aggressive IV fluid therapy and pain control are of more diagnostic value, because blood work should improve with just treatment alone), especially in sick, uremic cats, may include:
    - PCV/TS
    - BUN and creatinine
    - Serum sodium, potassium, and ionized calcium
    - Acid-base parameters
- Remove the catheter within 12 to 48 hours depending on patient stability and the gross appearance of the urine (eg, free of blood clots, isosthenuric).
- Monitor for at least 6 to 12 hours to ensure normal urination.
- Monitor for postobstructive diuresis:
  - Almost 50% of cats develop massive increases in their UOP following FUO, a phenomenon called postobstructive diuresis.
  - Aggressive IV fluid therapy is usually necessary in these cases to keep up with the losses and prevent dehydration, and even hypovolemia.
  - Typical fluid rates are 40 to 60 mL/h for patients with FUO to counter this severe diuresis.

### Table 6

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose</th>
<th>Comments</th>
</tr>
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<tbody>
<tr>
<td>Calcium gluconate 10%</td>
<td>0.5–1 mL/kg (give in mEq also) IV, slowly over 5–15 min; can be given as a faster bolus in patients that are periarrest</td>
<td>Treatment of choice for hyperkalemia, especially in animals that present with concurrently low ionized calcium levels. The ECG should be monitored closely throughout administration of this drug. This drug does not directly reduce potassium levels; instead it modifies the threshold potential, thereby offsetting the change in resting membrane potential caused by hyperkalemia and can be cardioprotective</td>
</tr>
<tr>
<td>Regular insulin</td>
<td>0.1–0.25 U/kg IV followed immediately with a 50% dextrose bolus (dose: 0.25–0.5 g/kg, IV, diluted 1:3)</td>
<td>Any IV fluid CRIs started subsequently are usually supplemented with between 2.5%–5% dextrose and blood glucose levels should be monitored closely over the next several hours to prevent hypoglycemia</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>1 mEq/kg slow IV, over 10–15 min</td>
<td>Typically used to treat refractory hyperkalemia that has not responded to the above 2 therapies. Use of sodium bicarbonate in the face of ionized hypocalcemia can further exacerbate hypocalcemia and increase the risk of clinical signs of hypocalcemic tetany and/or seizures. Therefore, calcium levels should be monitored closely. However, it may also be beneficial with severe metabolic acidosis</td>
</tr>
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</table>
As azotemia and dehydration resolve (as shown by isosthenuria and appropriate hemodilution of the patient), taper fluids to help reestablish a medullary solute gradient, because prolonged aggressive fluid therapy and diuresis often result in a medullary washout and loss of urine concentrating ability.

- Monitor ins and outs to ensure appropriate hydration and diuresis.
- Therapeutic cystocentesis:
  - Cystocentesis can be performed in cases in which catheterization is unsuccessful after repeated attempts. This treatment may help relieve the high hydrostatic pressure on the wall of the urinary bladder and, in some cases, may also relieve back pressure on the FUO and facilitate catheterization. Risks include bladder rupture and subsequent uroperitoneum, especially in patients that have an extremely inflamed and friable bladder wall.\(^{34}\)

The prognosis for survival to discharge in most cats with FUO is good, even in critically ill cats, providing they are stabilized within the first few hours of presentation. A study from 2008 reported a guarded long-term prognosis regarding reobstruction in these cats, with more than 50% of cats in the study showing recurrence of lower urinary tract signs within a year, and more than 30% of the cats having a repeat obstruction within the next 2 years.\(^{41}\) Perineal urethrostomy (PU) may need to be performed in cats that have repeated episodes of FUO, or are unable to be catheterized to relieve the obstruction. Client education is important in cases of FUO and feline lower urinary tract disease. Improved husbandry practices such as diet changes and switching to wet food, increasing water intake (eg, water fountain, grueled canned food), and environmental modifications (eg, kitty litter husbandry, including frequent, daily cleaning of litter boxes; use of favorable litter sources; and increased number of litter boxes) can all be instituted to help reduce the risk of reobstruction in these cats.

### Urethral Calculi in Dogs

- Canine urethral obstruction is another commonly seen urinary emergency in small animal practice. Urethral obstruction in dogs can be caused by urethral calculi, urethral strictures, or urethral neoplasia.
- Managing an acute urethral obstruction in a dog is similar to managing a case of FUO. After initial cardiovascular stabilization and correction of life-threatening electrolyte or acid-base abnormalities (eg, hyperkalemia, metabolic acidosis), relieving the urethral obstruction is the next step.

<table>
<thead>
<tr>
<th>Drugs</th>
<th>Doses</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzodiazepine such as midazolam or diazepam + ketamine</td>
<td>Midazolam/diazepam: 0.1–0.5 mg/kg IV  Ketamine: 0.5–4 mg/kg IV</td>
<td>Should be avoided if cardiac abnormalities are present, or if underlying cardiac disease is suspected</td>
</tr>
<tr>
<td>Benzodiazepine such as midazolam or diazepam + opioids</td>
<td>Midazolam/diazepam: 0.1–0.8 mg/kg IV  Opioids:  • Butorphanol (0.1–0.8 mg/kg)  • Methadone (0.1–0.4 mg/kg)  • Oxymorphone (0.02–0.05 mg/kg)</td>
<td>Safer to use in cats with preexisting heart disease or ECG abnormalities on presentation</td>
</tr>
</tbody>
</table>
Employ sterile technique.

Positioning: dorsal recumbency.

Clip and clean the area surrounding the penis and prepuce with a dilute antiseptic solution such as chlorhexidine.

Start catheterization using an open-ended tom cat catheter.

Lubricate the tip of the catheter with sterile lubricant gel.

Extrude the tip of the penis slightly past the prepuce.

Feed the catheter into the tip of the penis through the external urethral opening.

The penis can be allowed to slip back into the prepuce once the catheter tip is well seated inside the urethra.

Hold the prepuce and retract and direct it dorsally and caudally towards the tail base with the catheter still inside the tip. This:

- Straightens the urethra
- Facilitates easier dislodgement of the obstruction
- Facilitates easier passage of the catheter into the bladder.

Advance the catheter gently into the urethral passage while flushing aggressively to help alleviate the obstruction. Retract slightly if necessary and repeat flushing and advancing.

- Avoid excessive amounts of force while passing the catheter, and do not attempt to force it past an obstruction. The urethra in these cases is often extremely inflamed and friable, and can tear or rupture easily.

Fig. 5. Protocol for relief of FUO.
Use of urethral occlusion (urohydropulsion) while performing retrograde flushing to dislodge stubborn obstructions:

- Occlude the urethra around the catheter shaft using the thumb and forefinger while an assistant maintains pressure on the flush syringe to dilate the urethra by preventing reflux of the flush solution, and flush the obstructive material back into the urinary bladder.

Once the obstruction is relieved, pass the catheter further into the bladder until urine can be aspirated back.

Empty the bladder and flush several times with sterile saline until a clear solution is aspirated back. This helps remove as much grit and debris from the bladder as possible.

Leave an indwelling catheter in place, especially in cats that are uremic and critically ill, cats that have been obstructed for a long time, cats with detrusor atony, cats with possible urethral tear, or in cases in which there is significant amount of debris, grit, or blood clots in the urine that may cause a reobstruction.

- Typically, a 3.5-Fr or 5-Fr red rubber catheter is placed as an indwelling catheter and sutured in place using tape wings. This catheter is then connected to a sterile closed collection system to facilitate monitoring of urinary output and guide fluid therapy.

Fig. 5. (continued)

- Passing a urinary catheter (eg, red rubber, Foley) can help dislodge smooth-surfaced stones and push them back into the bladder until more definitive treatment of the calculi, such as a cystotomy or lithotripsy, can be performed.
- Urethral calculi typically tend to lodge just behind the caudal aspect of the os penis in dogs. The use of urohydropulsion may be necessary for emergency treatment of urethral obstruction caused by urethral calculi that cannot be easily dislodged by passage of a urinary catheter. Urohydropulsion is typically performed using a urinary catheter attached to a syringe filled with sterile saline (with or without a sterile lubricant mixed in equal proportions). The person performing the catheterization advances the urinary catheter into the urethral opening while an assistant maintains pressure on the flush syringe. The person performing the catheterization then occludes the urethra around the catheter shaft using the forefinger and the thumb. This technique helps dilate the urethra by preventing reflux of the flush solution, and helps flush the obstructive material back into the urinary bladder. Pulsatile pressure applied to the flush syringe may help dislodge the obstruction more quickly.
Fig. 6. Various types of urinary catheters for relief of FUO. From top to bottom: Mila™ urinary catheter, red rubber catheter, Tom Cat catheter.

Fig. 7. Positioning of patient for relief of FUO.

Fig. 8. Urinary catheterization in a male cat.
Clinical signs, management, and overall treatment are as discussed earlier for FUO.

Surgical removal of the calculi is typically required to prevent reoccurrence.

SUMMARY

Rapid recognition and aggressive therapeutic intervention is necessary with urogenital emergencies because of the potential to become life threatening if not addressed rapidly. Overall, the prognosis with urogenital emergencies is fair to good with appropriate medical or surgical management.

REFERENCES


Fig. 9. Indwelling catheter in a hospitalized cat following relief of FUO.