

INTERNAL MEDICINE

A Guide to Acute Kidney Injury in Small Animals

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Abstract

Diagnosis and management of acute kidney injury (AKI) require diligent attention to detail by the small animal clinician. The stepwise pathophysiology of AKI is often predictable. Common etiologies are categorized as prerenal, intrinsic, and postrenal. Diagnostic tools include palpation of the abdomen, kidneys, and urinary bladder; assessing hydration; monitoring blood pressure; urinalysis; and abdominal imaging and/or ultrasonography. Treatment involves fluid therapy, acid–base and electrolyte management, urine output and blood pressure optimization, nutritional support, and administration of medications as needed. Early use of extracorporeal therapy can improve outcomes. Prognosis varies according to the underlying causes, comorbidities, and prescribed treatments.

Take-Home Points

- Categories of acute kidney injury are prerenal, intrinsic, and postrenal; phases are initiation, extension, maintenance, and recovery.
- Diagnosis is made by palpation, assessment of hydration and blood pressure, urinalysis, abdominal imaging (radiography, ultrasonography), and sometimes testing for specific diseases.
- Treatment involves fluid therapy, acid–base and electrolyte management, urine output and blood pressure monitoring, and sometimes administration of antibiotics and/or other medications if needed.
- Prognosis for survival varies according to the etiology, ranging from 38% to 66%.

Acute kidney injury (AKI) is frequently recognized in veterinary practice, defined as an abrupt decrease in renal function with a corresponding decline in glomerular filtration rate (GFR).¹ Patients with community-acquired AKI (decreased kidney function occurring outside of a hospital setting, typically due to such issues as dehydration and urinary obstruction) usually present for evaluation days after the initial insult. As a result of increased monitoring and awareness, hospital-acquired AKI (decreased renal function during the course of hospitalization) is becoming more frequently recognized.²

Pathophysiology

The general types of injury leading to AKI have been categorized as prerenal, intrinsic (renal), or postrenal changes.

Prerenal

Prerenal azotemia is typically volume responsive. It involves decreased renal perfusion secondary to hypovolemia, intrarenal vasoconstriction, and decreased perfusion (secondary to shock).²

Intrinsic

Tubular damage is thought to occur via 1 of 2 mechanisms: hypoxic injury or direct nephrotoxic injury (following exposure to numerous drugs and

toxins).^{3,4} Intrinsic renal azotemia is subcategorized according to the type of injury.

- Vascular injury** (e.g., renal thrombosis) intuitively causes hypoxic injury to the kidney.
- Interstitial injury** is a broad category, encompassing infectious, inflammatory, and various other etiologies.
- Glomerular injury** involves some level of inflammation or other damage to the glomerulus; essentially, if the glomerulus is compromised, the remainder of the nephron will not function appropriately.

Postrenal

Postrenal azotemia involves obstruction to flow within the urinary tract (e.g., obstructive [ureteral, urethral]) or other mechanisms that prevent effective excretion of urine (e.g., urinary tract rupture).

Additionally, AKI encompasses 4 phases: initiation, extension, maintenance, and recovery.

Initiation

After some level of hypoxic insult, the initiation phase occurs immediately and is characterized by sublethal injury to tubular cells. The changes are secondary to breakdown of adenosine triphosphate (ATP), leading to cytoskeletal changes, loss of apical microvilli, loss of tight junctions between tubular cells, loss of attachment to the basement membrane, and

redistribution of sodium–potassium adenosine triphosphatase (Na-K-ATPase) pumps from the basolateral membrane to the apical membrane. These changes promote leakage of fluid and solute back into the interstitium, ultimately preventing normal function of the tubular cells.^{3,4} Sloughed tubular cells can accumulate in the tubules and are recognized as casts on urinalysis; their presence can cause an occlusive effect, further worsening GFR. During the initiation stage, biochemical and clinical abnormalities may not be appreciated.³

Extension

The extension phase is characterized by ongoing hypoxia combined with an inflammatory response. Necrotic and apoptotic tubular cells incite an inflammatory response in which neutrophils infiltrate first, followed by macrophages.³ Neutrophils will adhere to endothelial cells and migrate to the interstitium, plugging capillaries and altering renal tubular integrity. Continued ischemic injury and tubular cell death are perpetuated by blood stasis as well as release of cytokines, chemokines, and reactive oxygen species.³⁻⁵

Maintenance

During the maintenance phase, tubular cells undergo differentiation, migration, and proliferation⁴; azotemia and uremia are often present, and urine output may be excessively high or low. The maintenance stage can last days to weeks.

Recovery

During the recovery phase, redifferentiation continues and cells regain their polarity, allowing for normal cellular function. Trends toward polyuria during this phase result from retained solutes causing diuresis, and the kidney adapts to its new level of functionality.^{3,4} The recovery phase lasts weeks to months.

Etiologies

Potential causes of AKI are innumerable. Historical categorization into prerenal (volume responsive), intrinsic renal, and postrenal kidney injury remains useful when developing differential diagnoses.

Unfortunately, even with diligent diagnostic testing, the ultimate underlying cause of an AKI may not be found (up to 24% of patients in 1 study).⁶ Cardiorenal and hepatorenal syndromes are included among the different types of AKI etiologies in **BOX 1**; however, discussion of these syndromes is beyond the scope of this article.

Prerenal

Prerenal injury secondary to poor renal perfusion (hypovolemia) can be addressed by correcting intravascular volume deficits. Other causes, such as thromboses or poor perfusion secondary to systemic disease (e.g., heatstroke, disseminated intravascular coagulation) may be more difficult to detect and correct.

Intrinsic Renal

Intrinsic renal injuries include inflammatory, infectious, toxic, autoimmune, neoplastic, and various other general categories of insult (**BOX 1**).

Postrenal

Postrenal injury affects the urinary tract distal to the kidney; causes include ureteral obstructions, urethral obstructions, and uroabdomen.

Clinical Signs

Clinical signs of AKI in cats and dogs are often nonspecific. The clinical signs reported in the literature are usually anorexia, lethargy, tachypnea, weight loss, diarrhea, and vomiting.^{1,9,10}

Polyuria and polydipsia may be noticed, or they may go unnoticed if urinary habits are not strictly supervised. Oliguria and anuria are of concern in the presence of appropriate hydration. Renomegaly and pain may be noted during palpation of the kidneys but may not be noted for patients with preexisting chronic renal insufficiency (acute-on-chronic kidney injury).

Oral ulcerations can be encountered and may contribute to hyporexia. Tongue tip necrosis has been reported for uremic patients secondary to thrombosis. Coagulation profiles vary widely and include hypocoagulability; hypercoagulability (especially in

patients with glomerular disease); or a mix of normal, hypercoagulable, or hypocoagulable states.^{11,12}

Other organ systems can be affected in patients with AKI. One study reported that 79% of dogs with International Renal Interest Society (IRIS) grade III or higher AKI had documented ventricular premature contractions,¹³ likely secondary to myocardial injury as evidenced by cardiac troponin measurements. With respect to acute pancreatitis, more than 71% of dogs with AKI may exhibit increased lipase levels during hospitalization¹⁴; however, acute pancreatitis was subsequently confirmed for up to only 8.8% of dogs at admission and 14.9% during hospitalization.¹⁵ Uremic gastropathy reported in humans, for whom gastric ulceration is common, seems to be less common among dogs (in which mucosal edema and mineralization predominate)¹⁶; the same seems to be true for cats, in which gastric fibrosis and mineralization have been noted during histopathologic examination.^{17,18} Uremic encephalopathy may compound the clinical signs of lethargy and altered mental status via direct damage to white matter and astrocytes.¹⁹ Neurologic abnormalities and retinopathies have been reported with concurrent systemic hypertension; 16% of dogs with AKI at a referral hospital had evidence of retinal damage.²⁰ Specific diseases may also result in specific clinical

signs (e.g., pulmonary hemorrhage induced by leptospirosis).

Research models are finding more evidence of “renal crosstalk” and subsequent injury to distant organs secondary to the inflammation in patients with AKI.²¹ Future therapeutic targets have been suggested by documented hepatic, intestinal, pulmonary, brain, and cardiac involvement.²¹

Diagnosis

Thorough physical examination should include abdominal and renal palpation, urinary bladder palpation, and hydration assessment (xerostomia—that is, excessively dry mucous membranes despite appropriate hydration—may hinder hydration assessment). Blood pressure evaluation is imperative as a large number of patients are hypertensive at presentation or during hospitalization.^{20,22,23}

Establishing a standard minimum database is indicated for all AKI patients and should include a CBC, biochemical profile, and urinalysis (**BOX 2**). Venous blood gas analysis may help with further evaluation of acid–base status. If referral for extracorporeal therapy (e.g., dialysis) is a possibility, jugular venipuncture should be avoided.

BOX 1

Acute Kidney Injury Etiologies^{7,8}

Prerenal

- Hypovolemia
- Profound interstitial dehydration
- Cardiovascular renal disorders
- Hepatorenal syndrome
- Renal infarction
- Heatstroke

Intrinsic renal

■ Infectious

- Leptospirosis
- Lyme disease
- Babesiosis
- Pyelonephritis

■ Toxic

- Heavy metals (e.g., lead,

thallium, mercury, gold, nickel)

- Envenomation
- Carbon tetrachloride
- Vitamin D analogues (e.g., human dermatologic products)
- Cholecalciferol (e.g., rodenticides)
- Ethylene glycol
- Grapes/raisins (dogs)
- Lilies (cats)
- Hemoglobinuria/myoglobinuria
- Melamine

■ Other

- Neoplasia

- Iodinated contrast medium
- Drugs (e.g., aminoglycosides, amphotericin B, NSAIDs, cisplatin)
- Sepsis
- Acute pancreatitis
- Hyperviscosity syndromes (e.g., hyperglobulinemia, polycythemia)
- Ischemia

Postrenal

- Urethral obstruction
- Ureteral obstruction
- Urinary tract rupture (uroabdomen)

Urinalysis should ideally be performed via cystocentesis unless there are contraindications, such as coagulopathy. Despite the necessity of routine urinalysis, a study of dogs with AKI detected cylindruria and glucosuria (with normoglycemia) in only 31% and 23% of dog urine samples, respectively.²⁴ Urine should be submitted for culture and a urine protein:creatinine (UPC) ratio. Note that some studies have shown that an active sediment may confound the results of the UPC ratio, although the potential for proteinuria should not be ignored.²⁵

Abdominal radiographs (**FIGURE 1**) may be used to identify urolithiasis or other abnormalities in the lower or upper urinary tracts. Contrast cystourethrography or intravenous pyelography may help identify rupture or blockage of the lower and upper urinary tracts, although these procedures have been supplanted by more sophisticated techniques (e.g., computed tomography, fluoroscopy).

Abdominal ultrasonography is used extensively to evaluate the urinary system (**FIGURE 2**). In a study of cats with AKI, ultrasonography documented

renomegaly (68.9%), pyelectasia (57.8%), increased cortical (40%) and medullary (51%) echogenicity, and retroperitoneal fluid accumulation (33%).²⁶

Ultrasonographic guidance is recommended for fine-needle aspiration of the kidney (primarily for renal lymphoma) or core biopsies for patients with AKI or protein-losing nephropathies that require a definitive diagnosis.²⁷ Further evaluation of other intra-

BOX 2

Diagnostic Checklist for Acute Kidney Injury

Minimum database

- CBC
- Serum biochemistry
- Urinalysis
- Urine culture
- Abdominal radiography
- Blood pressure

Other considerations

- Abdominal ultrasonography
- Urine protein:creatinine ratio
- Urinary biomarker testing
- Venous/arterial blood gas measurement
- Specific disease testing (e.g., leptospirosis, ethylene glycol toxicity)
- Other imaging (e.g., fluoroscopy, computed tomography)
- Renal biopsy/fine-needle aspirates
- Coagulation testing (prothrombin time, activated partial thromboplastin time, viscoelastic testing)



FIGURE 1. (A) Ventrodorsal and (B) right lateral radiographs of a cat with acute kidney injury secondary to pyelonephritis. The right kidney (**asterisk**) is enlarged with decreased detail in that region, consistent with free fluid in the retroperitoneal space.

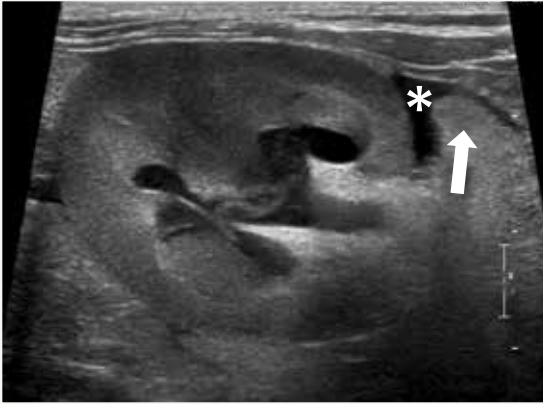


FIGURE 2. Sagittal ultrasound view of the right kidney of the same cat in **FIGURE 1**. There is ureteral dilation (marked by the crosses) as well as anechoic retroperitoneal effusion (**asterisk**) dissecting through hyperechoic perirenal fat (**arrow**).

abdominal organs may uncover other systemic diseases (e.g., sepsis, pancreatitis) contributing to AKI.

Specific disease states may require specific antidotes or antimicrobial therapy, according to history and index of suspicion. Ethylene glycol tests can detect the amount of toxin still free in the bloodstream. Leptospirosis diagnosis can be aided by point-of-care assays, although the mainstay of diagnosis remains microscopic agglutination serologic testing along with urine and blood PCR testing before starting treatment with antibiotics.²⁸

Several biomarkers have become more readily available and have shown utility for earlier detection compared with standard testing (e.g., serum creatinine).



RESOURCE 1

Scan the QR code to read "Clinical Application of Renal Biomarkers" from *Today's Veterinary Practice*.

Biomarkers include cystatin B,²⁹ NGAL (neutrophil gelatinase-associated lipocalin),³⁰ KIM-1 (kidney injury molecule-1), SDMA (symmetric dimethylarginine),³¹ and others. For a more detailed discussion of renal biomarkers, see **RESOURCE 1**.

Staging

In human and veterinary medicine, several iterations of illness severity scores for AKI have been adopted. In 2016, IRIS introduced AKI grading as a way to further characterize and stratify kidney disease; these guidelines closely mirror those of the human AKIN (Acute Kidney Injury Network). Injury severity is graded I through V, with subcategorizations of oliguric versus nonoliguric and whether the patient meets criteria for renal replacement therapy (RRT) (**TABLE 2**).

Of note, grade I AKI constitutes a >0.3 mg/dL increase in creatinine over baseline within 48 hours of a normal baseline reading or during hospitalization. Patients with this AKI grade can easily be overlooked because they are, by definition, nonazotemic (serum creatinine <1.6 mg/dL). Seemingly minor increases in creatinine are sometimes ignored when in fact the patient's status and response to treatment should be reevaluated because AKI has technically developed and the patient may be in the initiation stage of renal injury.

TABLE 2 International Renal Interest Society Acute Kidney Injury (AKI) Grading³²

GRADE*	SERUM CREATININE	NOTES
I	<1.6 mg/dL (<140 µmol/L)	Nonazotemic AKI is documented AKI and/or the following: - Progressive nonazotemic increase in creatinine of ≥0.3 mg/dL (26.4 µmol/L) in 48 hours - Measured oliguria (<1 mL/kg/hr urine output) or anuria over 6 hours
II	1.7–2.5 mg/dL (141–220 µmol/L)	
III	2.6–5 mg/dL (221–439 µmol/L)	
IV	5.1–10 mg/dL (440–880 µmol/L)	
V	>10 mg/dL (>880 µmol/L)	

*Each grade is substaged as nonoliguric or oligoanuric and/or requiring renal replacement therapy.

BOX 3**Summary of Acute Kidney Injury (AKI) Treatment Considerations**

- Reach a point of euvolemia via fluid therapy, avoiding hypervolemia.
- Perform serial physical examinations throughout the day, including hydration assessment and body weight measurements.
- Monitor electrolytes, ideally twice daily, and correct by altering IV fluid therapy or performing specific therapies (e.g., shift potassium intracellularly, extracorporeal therapy).
- Monitor blood pressure closely and treat hypertension. For patients experiencing an AKI crisis, angiotensin-converting enzyme inhibitors and angiotensin II receptor antagonists are not indicated.
- Administer antibiotics, ideally based on culture and susceptibility data, although empiric antibiotic use is reasonable while results are awaited. Antibiotics for specific disease states (e.g., leptospirosis) may need to be started before confirmation.
- Monitor urine output closely, ideally with a closed collection system. Plan to replace excessive fluid losses if the patient is polyuric, or consider medical therapies/extracorporeal therapy if the patient is oligoanuric.
- Treat nausea and vomiting.
- Address coagulation abnormalities, if indicated.
- Avoid any renotoxic drugs.
- Consider opioid analgesia if the patient is experiencing renal or other organ pain.
- Establish enteral nutrition as soon as possible, often with some type of feeding tube. In the patient's total fluid plan, account for fluid volumes in food administered. Phosphate binders can be administered when feeding.

Treatment

Treatment strategies vary according to the underlying etiology. However, several tenets of treatment require specific mention (**BOX 3**).

Fluid Therapy

Individualized IV fluid therapy is a mainstay of therapy for patients with AKI. The goals of fluid therapy are to reestablish intravascular volume, replace fluid deficits/losses, and meet maintenance fluid requirements (**BOX 4**). IV fluid therapy calculation should be based on estimated lean body weight, particularly for obese patients. Fluid therapy guidelines recently updated by the AAHA are directly applicable to patients with AKI.³³

Intravascular volume deficits, exhibited by signs of shock (e.g., tachycardia in dogs or bradycardia in cats, hypotension, prolonged capillary refill times, weak pulses) can be addressed with isotonic crystalloid fluid boluses equating to one-quarter to one-third of the patient's circulating blood volume over relatively short periods (15 to 30 minutes); if cardiovascular improvement is not noted, vital signs can be reevaluated and repeat administration can be considered.

Fluid deficits of the interstitial compartment are determined by typical signs of dehydration (e.g., loss of mucous membrane moisture, skin turgor, and body weight; globe retraction/corneal luster). These signs can be used to estimate a percentage of the patient's dehydration, which is then multiplied by the patient's body weight to provide an estimate of the fluid deficit in liters to be replaced over 6 to 24 hours.^{2,33} The fluid would ideally be a buffered isotonic crystalloid. The calculation of fluid needs in this manner is more precise than prescribing IV fluids at multiples of maintenance and corrects the patient's fluid deficits more efficiently. For a patient that is 5% dehydrated,

BOX 4**Fluid Therapy Goals**

- Reestablish intravascular volume (over 1–2 hours).
- Rehydrate the interstitial compartment (over 6–24 hours).
- Meet maintenance fluid needs.
- Replace ongoing fluid losses (insensible and sensible [measured]).
- Avoid overhydration/hypervolemia.

running twice-maintenance IV fluids may take 33 hours to fully rehydrate.³³

Numerous equations exist for calculating maintenance fluid requirements.³³ In many instances, balanced isotonic replacement crystalloid fluids are used and can be appropriate if electrolyte monitoring is available. Strictly speaking, hypotonic fluids (e.g., enteral free water, 0.45% NaCl, dextrose 5% in water [D5W]) should be used for maintenance needs because they provide the free water needed, whereas balanced isotonic crystalloids have sodium and chloride values that far exceed a patient's daily requirements.³⁴

Ongoing losses beyond maintenance, often resulting from polyuria, require replacement, typically provided via balanced isotonic crystalloids. The simplest method to quantify output is to frequently assess the body weight of a patient that has become appropriately hydrated (appropriate hydration of the interstitial compartment). At the very minimum, this assessment and a physical examination should be performed at least twice daily, if not more frequently. The losses exceeding maintenance needs can be added back into the fluid therapy plan. Ideally, patients with AKI would have indwelling urinary catheters connected to closed collection sets to enable precise evaluation of urinary output and losses that exceed what is being provided.

For patients with AKI, the ultimate goal is to establish a “zero fluid balance” (i.e., the input of fluid therapy matches the output after the intravascular and interstitial deficits have been corrected). IRIS working groups have recommended that hypovolemia be corrected within 1 to 2 hours of presentation and interstitial deficits be replaced within 6 hours, depending on patient tolerance.²

Historically, the concept of “forced diuresis,” involving challenging patients with IV fluids at multiples of the maintenance rate, has been a dogma of AKI therapy. However, acutely injured kidneys may not have the ability to handle these fluids and are at risk for fluid overload. Approximately 55% of patients in need of RRT are already fluid overloaded at arrival³⁵; anecdotal reports indicate that rates may be even higher. The kidney is an encapsulated organ, and increased interstitial hydrostatic pressure will cause decreased lymphatic outflow, peritubular capillary bed congestion, renin-angiotensin-aldosterone system

(RAAS) activation, increased inflammation, and ultimately decreased GFR.³⁶ Tubuloglomerular feedback from increased fluid load to the macula densa will in turn cause afferent arteriolar constriction, causing further decreases in GFR.³⁷ Thus, forced diuresis should be avoided for most patients.

Other organs will subsequently exhibit signs of dysfunction under conditions of fluid overload: intestinal edema can cause malabsorption and ileus, the heart may exhibit conduction disturbances or poor systolic and diastolic function, cholestasis results from hepatic congestion, poor wound healing and poor oxygen diffusion damage tissue beds, and pulmonary edema or pleural effusion can develop.³⁸ For veterinary patients with fluid overload, organ damage is perpetuated by the positive fluid balance, survival to discharge is worse, and the median duration of hospitalization is longer.³⁹

Acid–Base and Electrolyte Management

The normal homeostatic mechanisms for management of electrolytes are altered in patients with AKI.

Sodium

Sodium balance, most of which involves proximal tubular function, can be altered. Hypernatremia is usually associated with cats, through either chronic kidney disease or nonoliguric AKI.⁴⁰ Hypernatremia should be corrected slowly by administration of free water (e.g., D5W, enteral free water) at a rate not exceeding 0.5 mEq/L/hr; acute forms of hypernatremia can be corrected more quickly (1 mEq/L/hr).⁴¹ “Fluid creep,” wherein there is a slow gain of sodium from medication diluents, IV flushes, and other sources, should be avoided.

Potassium

Hyperkalemia is often considered an emergent concern in patients with AKI due to its effects on cardiac conduction. During a hyperkalemic crisis, serum potassium can be decreased to safer levels by shifting potassium into the intracellular space via administration of dextrose and regular insulin, taking advantage of their actions on cellular Na-K-ATPase pumps. Furthermore, inhaled β_2 agonists such as albuterol can stimulate the pump and also activate endogenous insulin release.⁴²

Sodium bicarbonate can be administered, although its use is typically reserved for patients that are extremely acidotic ($\text{pH} < 7.1$), are refractory to the above treatments, and have appropriate respiratory drive.^{2,43} Human trials involving sodium bicarbonate administration to patients have shown mixed results.⁴⁴

Administration of calcium salts (e.g., calcium gluconate) can reestablish the differences in resting membrane and action potentials in the heart, which serves as a temporary measure to stabilize arrhythmias while other interventions that actually lower serum potassium work. Calcium salts must be administered with caution to patients with hyperphosphatemia as increasing the calcium–phosphorus product may lead to dystrophic organ mineralization. Oral products such as sodium zirconium cyclosilicate have been used successfully in some patients that can tolerate oral medications.⁴⁵

Calcium

Ionized calcium can range from being low in patients with AKI to potentially being high (e.g., in patients with lymphoma). In patients with hypoalbuminemia, ionized calcium monitoring is ideal. Hypocalcemia can be addressed by careful administration of calcium salts with close monitoring. Hypercalcemia may require IV fluid therapy with 0.9% NaCl teamed with furosemide (in euvolemic patients) as well as corticosteroids eventually.

Phosphorus

Approximately 85% of feline patients with AKI are hyperphosphatemic⁴⁶; rates may be similar for dogs. First-line treatment consists of aluminum hydroxide to bind intestinal phosphorus, used after enteral nutrition has been initiated. Reported signs of aluminum toxicity secondary to aluminum hydroxide administration are dull mentation, ataxia, paresis, and hematologic abnormalities.⁴⁷ Other proprietary blends of aluminum- or calcium-based phosphate binders can be used as the patient becomes more stable.

Urine Output Management

Urine production is of the utmost importance for management of patients with AKI. Ideally, an indwelling urinary catheter is used to periodically measure urine output, although reasonable estimates can be obtained by monitoring serial body weights and

measuring urinations in other manners (e.g., weighing urine pads/bedding before and after voiding).

Oligoanuria, defined as urine output of $< 0.3 \text{ mL/kg/hr}$ over 6 hours in a euhydrated patient, is a concerning finding. Administering additional fluids to these patients will contribute to fluid overload and hypertension. Impending oligoanuria should raise concern if urine output is $< 1 \text{ mL/kg/hr}$ over 6 hours in the euhydrated patient.² Patients still undergoing rehydration will not produce normal amounts of urine; thus, oliguria and oligoanuria are declared only when the patient is fully hydrated.

For patients with oligoanuria that are at risk for fluid overload and for which extracorporeal therapies cannot be pursued, attempts should be made to convert the patient to a nonoliguric state. First-line drug therapy has traditionally been furosemide. Initially, 2 mg/kg IV can be administered; urine output results are usually noted in 20 to 40 minutes. Furosemide can be repeated hourly if needed. If there is no response, doses can be increased to 4 to 6 mg/kg IV ⁴⁸; dosages that lead to appropriate urine production can be administered every 6 to 8 hours.³ Other studies have investigated constant-rate infusions of furosemide with reported dosages of 0.66 mL/kg/hr ⁴⁹ to $0.7\text{--}1 \text{ mg/kg/hr}$ ⁷ after loading doses. Restoration of urine output does not imply improved GFR or outcome; appropriate urine output allows for continued fluid therapy to correct electrolyte and acid–base disturbances.³

Numerous other drugs have been investigated for their potential for reversing anuria. Mannitol will increase urine production and GFR after bolus injection; however, the results are short-lived and mild in dogs with normal renal function. Theoretically, mannitol has free-oxygen scavenging capabilities and may decrease renal tubular swelling.⁵⁰ However, the increased osmolality and concurrent risk for fluid overload make mannitol a potentially dangerous intervention for patients with AKI.² Dopaminergic agonists for patients with AKI have been investigated and have the theoretical benefit of decreasing afferent arteriolar tone and ultimately increasing GFR. Dopamine⁵¹ and fenoldopam⁵² administration have both failed to improve outcomes for patients with AKI. In an uncontrolled retrospective case series of dogs with AKI caused by leptospirosis, calcium channel blockers (e.g., diltiazem) showed some promise with

regard to renal recovery,⁵³ but investigations involving healthy dogs did not detect any improvements in GFR or urinary output after diltiazem administration.⁵⁴

Ultimately, during the recovery phase of AKI, profound polyuria (> 2 mL/kg/hr) may occur secondary to loss of concentrating ability, tubular injury, osmotic diuresis from retained solutes, and lack of responsiveness to antidiuretic hormone.⁵⁵ Again, balanced isotonic crystalloid replacement fluids can be administered to replace urine output that is in excess of maintenance fluid requirements. Polyuria can last for days during the recovery period (median hospitalization of 5 days).⁶ After blood urea nitrogen and creatinine levels have plateaued and fluid inputs and urinary outputs have equalized, IV fluids can be tapered slowly. A reasonable goal is a decrease of 15% to 20% every 8 hours, with a plan to discontinue fluids within 48 hours.³⁸ During this period, if the patient's urinary output is higher than what is being replaced, or if renal values increase, then IV fluids need to again be increased.

Blood Pressure Monitoring

Several mechanisms lead to hypertension in patients with AKI (e.g., RAAS activation, volume overload, sympathetic activation). The kidney is one of the target organs for damage by systemic hypertension. Regular monitoring of blood pressure in hospitalized patients is crucial; systemic hypertension develops in 58.7% of cats during hospitalization, severe (> 180 mm Hg) in 28.2%.²² The trend in dogs is similar; hypertension develops in 81%, severe in 62%.²³ Obtaining serial blood pressure measurements in hospitalized patients according to consensus guidelines,⁵⁶ as well as other ancillary diagnostics such as serial retinal examinations,²⁰ will help guide therapy. Typical initial treatment includes administration of calcium channel-blocking medications such as amlodipine to promote arterial dilation. Blood pressure reduction should be controlled, with a decrease of 10% in the first hour, followed by 15% over the next several hours.⁵⁷ α -Adrenergic blockade with acepromazine can be considered as a second- or third-line agent.² Other injectable medications such as sodium nitroprusside and hydralazine may have limited availability and ideally are titrated via invasive blood pressure monitoring. For patients with AKI, use of angiotensin-converting enzyme inhibitors and angiotensin II receptor blockers is contraindicated.

Antibiotic Therapy

Antibiotic use should be based on initial working diagnosis and eventual culture/susceptibility data. Suspected leptospirosis can be treated empirically with injectable β -lactams initially, followed by doxycycline to reduce organism persistence in the renal tissues. If serum creatinine is > 5 mg/dL or if the AKI is IRIS grade IV or higher, then doubling the dosing interval of some antibiotics is recommended.²⁸ Consensus guidelines recommend empiric therapies against Enterobacteriaceae for pyelonephritis (e.g., a fluoroquinolone, cefpodoxime); additional IV choices include cefotaxime or ceftazidime. A treatment course of 7 to 14 days may be sufficient.⁵⁸

For patients with urinary catheters in place, careful nursing care is mandatory. Patients with noninfectious disease, such as cats with urethral obstruction that have profoundly low rates of bacteriuria,⁵⁷ do not require antibiotics while catheterized unless showing systemic signs of infection/urosepsis. Cleansing of the exposed urinary catheter areas with dilute chlorhexidine will prevent biofilm accumulation.⁵⁹ If the patient shows evidence of cystitis, repeat culture via cystocentesis can be performed at the time of urinary catheter removal.⁵⁸

Other Medications

In human medicine, dosage adjustments for those with AKI are common as alterations in volume of distribution, protein-binding capabilities, oral bioavailability, and GFR cause the pharmacokinetics of many drugs to be unpredictable. Clinicians evaluate each drug administered, considering its normal half-life and safety profile/therapeutic window. Prolonging the dosing interval or dose reduction is performed frequently in human medicine; estimating GFR is also common.

In veterinary medicine, GFR quantification is not as thoroughly investigated nor typically evaluated; however, current guidelines recommend basing dosing alterations on rough estimates of GFR according to IRIS AKI grade. Specific recommendations are available in the IRIS AKI guidelines.² For instance, if a drug has a very narrow therapeutic index or can lead to toxicity in patients with decreased renal clearance, dose reduction or prolonged dosage interval may be indicated.

In patients with AKI, nausea and vomiting are common clinical signs. Centrally acting antiemetics such as maropitant are indicated, as are other drugs such as 5-HT₃ antagonists (e.g., ondansetron) or metoclopramide. Gastroprotectants (e.g., H₂ receptor antagonists, proton pump inhibitors) can be indicated if evidence of gastric ulceration is noted; however, no specific guidelines exist for patients with AKI. Consensus guidelines do not recommend prophylactic use of gastroprotectants in patients with chronic kidney disease.⁶⁰

Patients with protein-losing nephropathies, patients with thromboses, or those documented to be in a hypercoagulable state via viscoelastic testing may benefit from antithrombotic medications such as clopidogrel or anticoagulants (low-molecular-weight or unfractionated heparins). Drugs such as rivaroxaban are becoming more affordable and may represent more convenient and targeted anti-Xa activity.

Alternatively, hypocoagulation, resulting from platelet dysfunction (uremic thrombopathy), thrombocytopenia, or clotting factor consumption from disseminated intravascular coagulation, may require treatment with blood products.

For patients experiencing pain, typically abdominal or renal, pure μ agonists (e.g., fentanyl, hydromorphone, methadone) or other partial μ agonists (e.g., buprenorphine) can be administered, preferentially parenterally. The adverse effects of nausea and ileus should be monitored and treated. Use of NSAIDs in patients with AKI is absolutely contraindicated.

Specific therapies may be targeted, depending on the underlying disease process. For ethylene glycol ingestion, infusions of ethanol or 4-methylpyrazole or timely referral for dialysis are imperative for preventing renal injury. Other renal toxicities need to be treated promptly through standard decontamination if indicated (e.g., emesis, activated charcoal). Emerging therapies for toxicities include therapeutic plasma exchange as well as hemoperfusion.

Nutrition

Patients with AKI are known to be in a hypercatabolic state. One study found that despite being fed 1.3 times resting energy requirement, patients with AKI lost

1.16% of body weight per day.⁶¹ Patients will not be able to keep up with the metabolic demands of AKI via voluntary oral intake. Forced syringe feeding is not recommended due to food aversion and risk for aspiration pneumonia.² Early enteral nutrition should be implemented as soon as feasible. Nasogastric/nasoesophageal tubes are easy to place and maintain; however, the diet formulations are limited. Esophagostomy or gastrostomy tube placement requires that the patient be under general anesthesia; however, a wider variety of diets can be fed and the tubes can be maintained for a longer period.

Although protein restriction for patients with chronic renal disease is intuitive, it is not the focus for patients with AKI. Various recovery diets that reach normal protein requirements, ideally with restricted phosphorus, are recommended.⁶² Note that any volume of liquid recovery diets or water used to suspend blended foods for large-bore feeding tubes counts toward fluid intake and must be subtracted from the patient's total IV fluid administration.

Extracorporeal Therapy

Once considered a modality only in human medicine, in the past several years the number of veterinary regional referral centers offering extracorporeal therapy (e.g., continuous RRT, intermittent hemodialysis, hemoperfusion, therapeutic plasma exchange) to address AKI has increased. Although often costly, early consideration of these modalities can prolong the window available for renal recovery, ultimately improving outcome. Treatments can manage electrolyte disturbances, remove excess fluid through ultrafiltration, and take over the excretory functions of the diseased kidney. Consensus guidelines on when to consider intermittent hemodialysis are summarized in **BOX 5**.⁶³

Prognosis

Prognosis for patients with AKI varies greatly; survival rates range from 38%⁶⁴ to 66%⁶; prognoses for patients undergoing intermittent hemodialysis fall within this range.⁶⁵ Veterinary studies are inherently affected by euthanasia bias.

Etiology significantly affects survival rates. The survival rate for dogs with AKI secondary to ethylene

glycol toxicosis is roughly 12% after diagnosis, and the rate for cats is 8%.⁶⁶ The survival rate for cats with AKI after lily toxicosis is as high as 87.5% to 100%, although these values are affected by the timing of presentation after lily exposure and the treatments.⁶⁷ The survival rate for dogs in which severe AKI developed after grape ingestion was 53%.⁶⁸ The survival rate for cats with ureteral obstruction relieved with subcutaneous ureteral bypass can be approximately 90%.⁶⁹ For cats with pyelonephritis, the rate is 57%.⁴⁶

The outcome for dogs with leptospirosis can be reasonable (68% survival rate), although this rate is influenced by extrarenal injury (e.g., pulmonary hemorrhage, hepatic involvement).¹⁰ Another study in which 12/14 dogs were treated with intermittent hemodialysis and 18/22 were treated conservatively found a survival rate of 83%.³⁵

Acute-on-chronic kidney disease in cats resulted in a survival rate of 58%; acute-on-chronic kidney disease in dogs resulted in a survival rate of 65%.¹ However, for both populations, the long-term survival was guarded.

Patients with volume-responsive (i.e., prerenal) AKI survived 82.7% of the time, whereas patients with intrinsic AKI survived 47.8% of the time.⁷⁰

Several researchers have investigated prognostic indicators for AKI. Of note, AKI grade is not necessarily associated with outcome, emphasizing the reversibility and recovery associated with kidney injury.⁷¹ Among

other abnormalities, nonsurvival has been associated with anemia, thrombocytopenia, anuria, hyperkalemia, hypoalbuminemia, hypocalcemia, proteinuria, and elevated anion gap, depending upon the underlying cause.^{6,8,24,61}

At the initial consultation, clients should be informed of the involved case management, survivability to hospital discharge, and eventual long-term management. The prognosis percentages are specific to populations of patients with AKI of varying causes, but individual patients vary, especially when the entirety of the clinical picture and comorbidities are accounted for. Creatinine may normalize completely for 55% of patients with AKI by discharge and for a total of 75% by the next recheck.⁷¹ Clients should be prepared for the potential need for long-term management for a patient with chronic kidney disease after recovery from AKI. One study showed a median survival time of 1322 days, although this time is not necessarily directly applicable to individual patients with AKI of widely varying etiologies and their respective mortality rates.⁷¹

Summary

AKI in cats and dogs has innumerable underlying causes, which can be prerenal, renal, or postrenal. Patients often exhibit nonspecific clinical signs; however, obtaining a thorough history and performing complete diagnostics may help further characterize the underlying cause. Treatment during renal recovery is primarily supportive and includes targeted fluid therapy, with an emphasis on preventing fluid overload, careful management of electrolytes, monitoring urine output and blood pressure, providing enteral nutrition, and administering antibiotics and antidotes as necessary. Prognosis varies greatly based on the underlying cause, treatment modalities chosen, and comorbidities. **TVP**

BOX 5

Potential Indications for Intermittent Hemodialysis⁶³

- Serum creatinine >5 mg/dL (442 µmol/L) and trending higher in an appropriately hydrated animal
- Persistent (> 6 hr) anuria or oliguria (<0.3 mL/kg/hr urine output) unresponsive to appropriate fluid therapy and medical management
- Severe/life-threatening fluid overload not responsive to diuretics
- Electrolyte disturbances that cannot be controlled with standard medical therapy

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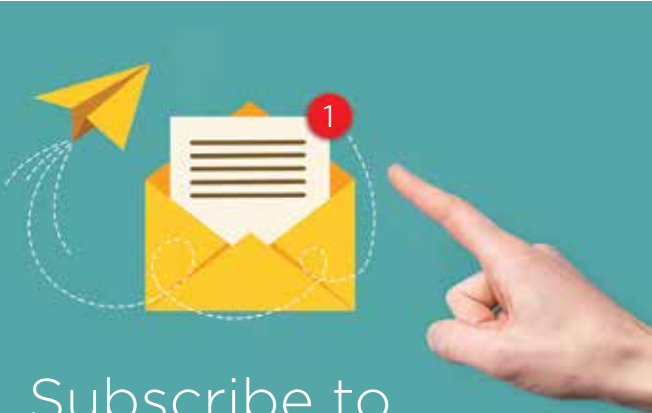
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CE Quiz Questions

1. **Which underlying disease/condition has not been associated with the development of acute kidney injury (AKI)?**
 - a. Aminoglycoside antibiotics
 - b. Leptospirosis
 - c. Uveitis
 - d. Fluid overload
2. **A 5-kg (11-lb) cat has a creatinine level of 4.5 mg/dL and normal acid–base parameters, shows no evidence of hypertension or hypotension, and appears to be euhydrated. Imaging and initial diagnostics are consistent with pyelonephritis. What is the best fluid plan for this cat?**
 - a. An individualized plan that accounts for maintenance needs and balanced isotonic crystalloids that replace urinary outputs beyond maintenance requirements
 - b. Twice maintenance 0.9% NaCl until the cat goes home
 - c. Two to 3 times maintenance lactated Ringer's solution to force diuresis and drive kidney values down
 - d. No IV fluids; the cat should drink enough on its own.
3. **Patients with an indwelling urinary catheter should receive antibiotics, regardless of the cause of their AKI.**
 - a. True
 - b. False
4. **A dog has a creatinine of 5.1 mg/dL. After 24 hours of hospitalization and IV fluids, the patient has gained 10% of its body weight and has produced 0.2 mL/kg/hr in the urinary collection set. At recheck, the creatinine is now 6.2 mg/dL. What is the most appropriate action in this case?**
 - a. Increase the IV fluid rate because the higher fluid rate will help drive renal values down and produce more urine.
 - b. Continue with the same IV fluid therapy plan; depriving the dog of IV fluids will cause the creatinine to increase.
 - c. Medically manage the oliguria with an injectable dose of furosemide and temporarily stop IV fluids.
 - d. Place a nasogastric tube to provide enteral water only.
5. **Which drug has not been shown to decrease potassium in a hyperkalemic crisis?**
 - a. Albuterol
 - b. Dextrose and regular insulin
 - c. Calcium gluconate
 - d. Sodium bicarbonate



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