Acute renal failure (ARF) is due to rapid hemodynamic, filtration, tubulo-interstitial, or excretory injury to the kidneys, the outflow tract, or both. The abrupt decrease in glomerular filtration rate (GFR) results in accumulation of uremic toxins and metabolic wastes, metabolic dysfunction, and dysregulation of fluid, electrolyte, and acid–base balance. The kidneys are particularly susceptible to ischemic and toxic damage because they receive 20% of cardiac output. Certain medications are concentrated in the kidneys secondary to tubular secretion or reabsorption, which can predispose patients to renal injury. ARF can develop because of prerenal, intrinsic renal, or postrenal causes.

Prerenal disease develops when the GFR declines because of decreased renal blood flow or increased renal vascular resistance. When the mean arterial pressure is less than 70 to 80 mm Hg, renal perfusion is compromised. Prerenal azotemia is a functional abnormality that is potentially reversible. Prerenal disease can coincide with intrinsic renal failure, and prolonged prerenal azotemia can progress to structural damage and intrinsic, irreversible renal failure. Many causes of prerenal azotemia are avoidable and/or easily treated before subjecting patients to therapies that may predispose them to ARF. Dehydrated patients should be rehydrated before general anesthesia or administration of potentially nephrotoxic substances. In humans, volume depletion leads to a tenfold increased risk of developing ARF. Hypotension during anesthesia and surgery can be avoided by administering intravenous fluids and closely monitoring blood pressure during medical procedures.

Intrinsic causes of ARF result from renal parenchymal damage due to ischemic, glomerular, or tubular disease. In dogs and cats, toxic, infectious, and ischemic causes occur most commonly. Postrenal causes are due to obstruction or rupture of the urinary tract. Obstruction may occur from ureoliths, neoplasia, trauma, inflammation, fibrosis, blood or mucus, and congenital or acquired ureteral stenosis. If postrenal ARF is recognized and treated early, most cases are reversible. Postrenal disease lasting longer than 7 days can cause renal parenchymal damage and ARF.
Medications associated with the induction of ARF are listed in the box above.

**Antimicrobials**
- Aminoglycosides
- Carbenems
- Fluoroquinolones
- Rifampin
- Tetracyclines
- Aminoglycosides
- Amphotericin B
- Cephalosporins
- Penicillins
- Sulfonamides
- Vancomycin

**Cancer chemotherapy**
- Bisphosphonates
- Cisplatin
- Methotrexate
- Carbohtepin
- Doxorubicin

**Immunosuppressants**
- Azathioprine
- Cyclosporine

**Other medications**
- Allopurinol
- Apomorphine
- Dextran-40
- NSAIDs
- Penicillamine
- Angiotensin-converting enzyme inhibitors
- Cimetidine
- Mannitol
- Streptokinase

**Risk Factors for Acute Renal Failure**
- Preexisting renal disease
- Decreased cardiac output
- Dehydration
- Hypomagnesemia
- Advanced age
- Hypocalcemia
- Hypotension
- Hypokalemia
- Fever
- Hyponatremia
- Sepsis
- Acidosis
- Hepatic insufficiency
- Concurrent use of potential nephrotoxins
- Trauma
- Pancreatitis
- Diabetes mellitus
- Systemic hypertension
- Hypoalbuninemia
- Anesthesia and surgery
- Hypoadrenocorticism
- Vasculitis
- Trauma
- Disseminated intravascular coagulation
- Heatstroke
- Hyperviscosity syndrome (multiple myeloma, polycythemia)

**Antimicrobials**
Many antibiotics are potentially nephrotoxic to dogs and cats. The nephrotoxic potential of these drugs increases in animals with risk factors for ARF, including dehydration, hypotension, general anesthesia and surgery, and certain metabolic conditions. Additional risk factors are listed in the box to the right. Prophylactic nafcillin administration to prevent perioperative infection was associated with postoperative ARF in seven dogs with no history of preexisting renal disease. Aminoglycosides are among the most nephrotoxic antimicrobials, and their systemic use in animals has been associated with ARF for decades. Few cases of aminoglycoside-induced ARF have been reported since the 1980s, although there are two recent case reports involving cats. One cat developed fatal ARF within 4 days of topical administration of gentamicin to a soft tissue wound during lavage. A total dose of 232 mg/kg was applied. The serum gentamicin level was greater than five times therapeutic concentrations. Four cats administered paromomycin for treatment of large bowel diarrhea due to enteric trichomoniasis or cryptosporidiosis developed ARF. Paromomycin is an orally administered aminoglycoside that is poorly absorbed from the gastrointestinal (GI) tract. The dose administered in the four cats ranged from 70 to 208 mg/kg PO q12h for fewer than 5 days; previous studies showed no toxicity at doses of 125 to 165 mg/kg PO q12h for 5 days. All cats developed lethargy, vomiting, anorexia, dehydration, and azotemia within 4 days after therapy was initiated. Azotemia improved or resolved within 6 to 11 months in all cats. The cats may have been at a higher risk of developing renal toxicosis partly because of increased systemic absorption of paromomycin from diseased bowel.

**NSAIDs**
Nephrotoxicity due to administration of NSAIDs, including selective cyclooxygenase (COX) inhibitors, is most common in patients with concurrent renal disease, patients with conditions predisposing them to renal damage, or those that have consumed greater-than-therapeutic doses. NSAIDs can decrease renal perfusion because prostaglandins (PGs) E2 and I2 mediate afferent arteriolar dilation in response to decreased renal
blood flow and PGI2 stimulates renin release. Most studies in veterinary medicine have been conducted on healthy patients undergoing elective procedures. Carprofen did not decrease the GFR (based on renal scintigraphy) or cause histopathologic changes in the kidneys when administered to healthy dogs. Ketoprofen has been associated with transient azotemia in healthy dogs undergoing ovariohysterectomy. 

All NSAIDs represent a risk for renal function, although not equally. Selective COX-2 inhibitors are reportedly safer than nonselective NSAIDs in humans and cause fewer GI and renal side effects. Both types of NSAIDs are potentially toxic in dogs and cats, particularly because of species differences in the expression and function of COX isoforms. There are two isoforms of COX: COX-1 is expressed constitutively and is involved in the production of PGs needed for normal physiologic functions in several organs, including the kidneys. COX-2 expression is inducible by bacterial endotoxins, cytokines, and growth factors but is also expressed constitutively in the kidneys in the absence of inflammatory stimulation. COX-2 expression is greatly increased in dogs that are volume or sodium depleted; therefore, administration of NSAIDs that preferentially inhibit COX-2 may cause renal damage, particularly in dehydrated patients.

Renal disease in dogs has been associated with administration of aspirin, carprofen, flunixin meglumine, naproxen, and phenylbutazone; however, ibuprofen is the most common generic drug generating calls to the National Animal Poison Control Center regarding dogs and cats. Appropriate intervention in cases of ibuprofen-induced disease was more effective in managing GI disease than in preventing renal disease. ARF generally occurred 36 hours or more after exposure, whereas GI signs occurred within a few hours. In a research setting, an ibuprofen dose of 300 mg/kg was required to induce ARF in dogs, whereas clinical disease has occurred with consumption of 200 mg/kg or less. Cats are susceptible to ibuprofen doses approximately one-half of those reported as toxic in dogs.

NSAID use should be avoided in patients at high risk of developing renal disease, including dehydrated patients and those with cardiac or renal disease. Patients undergoing general anesthesia and perioperative NSAID administration should be well hydrated before anesthesia and should receive fluid support to maintain normal blood pressure. Most dogs that develop NSAID-induced ARF have a favorable prognosis and generally respond well after appropriate treatment for 5 to 10 days.

**TOXICANTS**

**Ethylene Glycol**

Ethylene glycol is a common cause of fatal poisoning and ARF among dogs in the United States, with an estimated 10,000 to 45,000 cases occurring annually. Because antifreeze is a widely available source of ethylene glycol, cases most often occur during seasons in which antifreeze is used. Ethylene glycol is also present in industrial solvents, rust removers, color film processing fluids, and heat-exchange fluids.

Ethylene glycol is absorbed quickly from the GI tract. Blood concentration peaks within 1 to 4 hours after ingestion, and almost all of the toxin is metabolized or excreted within 18 to 24 hours after ingestion. Ethylene glycol initially crosses the blood–brain barrier, where it exerts narcotic or euphoric effects similar to those of ethanol. The second clinical stage occurs when ethylene glycol is metabolized to acidic intermediates, including glycolic, glyoxylic, and oxalic acid, which cause severe metabolic acidosis; this occurs within 3 to 4 hours after ingestion. Oxalate binds to plasma calcium, forming calcium oxalate crystals in the renal tubules (Figures 1 and 2). Signs of cardiopulmonary disease may also develop.

Two proposed mechanisms for renal tubular damage include formation of calcium oxalate crystals in blood vessels and renal tubules and direct nephrotoxic effects of the metabolites of ethylene glycol. Animals that develop ARF frequently show signs of severe depression, become comatose, and die. Lethal doses of 95% ethylene glycol in cats and dogs are 1.4 to 4 ml/kg and 4 to 6.6 ml/kg, respectively. Antifreeze–induced ARF might become less frequent with the use of brands that contain propylene glycol, which is less toxic, or a bitter aversive agent (denatonium benzoate).

**Vitamin D**

Vitamin D intoxication is well described in dogs and cats. The most common source is ingestion of rodenticides containing cholecalciferol (vitamin D3), which is available in a variety of formulations and brand names. In addition, toxicosis can occur from oversupplementation or feeding diets containing excessive vitamin D. The major pathophysiologic effect is hypercalcemia, which can cause ARF. Acute renal tubular damage occurs as a result of altered cell membrane permeability, altered calcium pump activity, decreased cellular energy production, and cellular necrosis. The toxic dose in naturally exposed dogs is 1.5 to 8 mg/kg, and the
median lethal dose is 13 mg/kg in adult dogs.\textsuperscript{16,17} The toxic dose is unknown in cats. In general, puppies are more susceptible to vitamin D toxicosis than are adult dogs, and cats are more sensitive than dogs.\textsuperscript{17}

Vitamin D toxicosis can occur with ingestion of human medications containing vitamin D as treatment of hypophosphatemic disorders, hypoparathyroidism, osteomalacia, osteoporosis, and renal failure. An additional vitamin D toxicant is calcipotriene, which is a synthetic analogue of calcitriol. Calcipotriene is the active ingredient in Dovonex (Bristol-Myers Squibb), a topical medication used to treat psoriasis in humans.\textsuperscript{18}

Ingestion of calcipotriene can cause severe hypercalcemia, ARF, soft tissue mineralization, and death in dogs.\textsuperscript{18,19} Within 12 to 24 hours after ingestion, dogs exhibit vomiting, depression, anorexia, polyuria, and diarrhea.\textsuperscript{18,19} Hypercalcemia, hyperphosphatemia, and hypercalcemic nephropathy can occur within 18 to 72 hours; the animal can die from calcification of cardiac tissue weeks after ingestion and resolution of acute clinical signs and/or laboratory abnormalities.\textsuperscript{18,19}

**PLANTS**

**Raisins and Grapes**

Since 2001, acute GI and renal toxicity has been reported several times in dogs after ingestion of raisins or grapes. Most cases have occurred since 1999 in both the United States and United Kingdom.\textsuperscript{20,21} Affected dogs consumed organic or commercial grapes, both red and white varieties, as well as crushed or fermented grapes from wineries. Raisins were mostly commercial sun-dried varieties of various brands.\textsuperscript{20} Estimated amounts of ingested raisins or grapes ranged from 3 to 57 g/kg.\textsuperscript{20-23} A specific toxicant has not been identified. Contamination of grapes and raisins with insecticides, pesticides, heavy metals, or mycotoxins has been proposed and investigated but not proven. There are no known reports of raisin or grape toxicity in other species.

Initially, clinical signs of vomiting and lethargy develop within hours after ingestion. Anorexia, diarrhea, and abdominal pain have also been reported. ARF can develop within 24 to 72 hours after ingestion of raisins or grapes. Dogs may become oliguric or anuric. Death in dogs with ARF secondary to raisin or grape ingestion has occurred in 50% to 75% of reported cases.\textsuperscript{20,21,23} All dogs that recovered were managed aggressively, including the use of peritoneal dialysis in some cases.\textsuperscript{23} Histopathologic findings included acute proximal renal tubular degeneration or necrosis, metastatic mineralization of numerous tissues, frequent tubular casts, intact basement membranes, and evidence of renal tubular epithelial regeneration.\textsuperscript{22,24} In one report\textsuperscript{22} of 10 dogs with ARF associated with grapes or raisins, a golden-brown globular pigment was observed in the renal tubular epithelial cells in six cases; its significance is unknown.

**Lilies**

Ingestion of members of the genera *Lilium* (Easter lily, tiger lily, stargazer lily, Asiatic hybrid lily) and *Hemerocallis* (common daylily [Figure 3], early daylily)
has been associated with ARF in cats. Other plants commonly referred to as lilies (e.g., calla lily, peace lily) are not nephrotoxic. The toxic principle in Easter lilies is present in aqueous extracts of both flowers and leaves, although flowers are more toxic. Doses equivalent to one flower or eight leaves induced clinical toxicosis in cats exposed in a research setting. The toxic principle and dose are unknown for other lily species.

Lily toxicosis initially causes vomiting and ptyalism after plant ingestion. Neurologic signs (e.g., ataxia, depression, tremors and seizures, ARF) can also develop. Renomegaly, renal pain, oliguria, and anuria have been reported. In one report, cats diagnosed with ARF developed severe azotemia 2 to 5 days after ingestion (mean serum urea nitrogen: 215 mg/dl [normal: 15 to 34 mg/dl]; mean serum creatinine: 22.3 mg/dl [normal: 0.8 to 2.3 mg/dl]). Cylindruria, glucosuria, and proteinuria have been documented.

Mortality rates of 50% to 100% have been reported in cats developing ARF. There are no histopathologic reports in daylily toxicosis cases, but histopathologic changes in the kidneys of cats that have ingested Lilium plants include acute renal tubular necrosis, interstitial edema, polarized crystals in the collecting tubules, and renal tubule epithelial cell regeneration. Although the toxic agent can cause severe regional destruction of renal tubular epithelial cells, basement membranes are usually intact, thereby allowing possible tubular regeneration.

**INFECTIOUS DISEASE**

**Leptospirosis**

Leptospirosis is a worldwide zoonotic disease affecting numerous species, including humans, dogs, and livestock. It is caused by spirochete bacteria and has been reported in dogs in the United States and Canada for more than 100 years. There are more than 220 pathogenic leptospiral serovars and many more that are avirulent. Pathogenic serovars are divided into three species:

- *Leptospira interrogans*—serovars Canicola, Icterohaemorrhagiae, Bratislava, Hardjo, Pomona, Australis, and Autumnalis
- *Leptospira kirschneri*—serovar Grippotyphosa
- *Leptospira borgpetersenii*—serovar Ballum

Serovars are maintained in the environment by one or more reservoir hosts in which infection is typically asymptomatic. Dogs are a reservoir host for the serovar Canicola but also serve as secondary hosts for other serovars that cause illness. Leptospiral infection can be asymptomatic or cause ARF, hepatic disease, coagulation disorders, or a combination of syndromes.

Historically, most cases of canine leptospirosis were caused by serovars Canicola and Icterohaemorrhagiae, but recent clinical reports have shown that the disease is now most commonly associated with serovars Grippotyphosa, Pomona, Autumnalis, and Bratislava. The change in the epidemiology of canine leptospirosis may be due to urbanization leading to increased exposure of dogs to wildlife and livestock reservoir hosts. In addition, there has been a marked decrease in disease caused by serovars Canicola and Icterohaemorrhagiae since widespread vaccination of dogs beginning in the 1970s.

Studies have found that male dogs, dogs 4 years of age or older, herding dogs, hounds, and working dogs are at a significantly higher risk of developing this disease. Despite these risk factors, leptospirosis is also diagnosed in toy breeds and young dogs, and it is believed that...
German shepherds may be predisposed. There has also been an association between increased rainfall and increased numbers of cases of canine leptospirosis. Most cases of clinical leptospirosis in dogs in the past two decades have manifested as ARF with or without hepatic involvement (Figure 5). Affected animals rarely present with hepatic disease as the only clinical problem.

Once the organism penetrates mucous membranes or abraded skin, it replicates in the vasculature and disseminates to the kidneys, where it enters the interstitium. Organisms can be seen within tubular cells and tubular lumen within 2 weeks after infection. After colonizing renal tissue, the organism causes tubulointerstitial nephritis with interstitial edema and cellular infiltrate, which can lead to swelling and impaired renal perfusion. Leptospiral organisms have several pathogenic factors that are nephrotoxic. Leptospiral lipopolysaccharide (LPS) and other outer-membrane components damage cells. Leptospiral LPS is a potent macrophage activator that stimulates secretion of interleukin-1 and interferon, augmenting macrophage killing capacity. In vitro, the LPS causes platelet aggregation, lysis, and degeneration and stimulates release of tumor necrosis factor-α and interleukin-10 by human peripheral blood mononuclear cells. These cytokines may play a significant role in the inflammatory response to leptospiral organisms.

With early recognition, appropriate antibiotic therapy, aggressive fluid therapy, and perhaps dialysis, many dogs survive infection, although a mortality rate of 11% to 33% has been reported. Serovar Pomona has been associated with a worse prognosis than have other serovars.

**Pyelonephritis**

ARF due to pyelonephritis most commonly occurs secondary to ascending lower urinary tract infection caused by naturally occurring bacterial cystitis or nosocomial transmission via urinary catheterization. Hematogenous spread of bacteria to the kidneys can also occur. Conditions associated with hematogenous spread include bacterial endocarditis, diskospondylitis, and pyometra, whereas chronic bacterial cystitis, hyperadrenocorticism, and diabetes mellitus predispose patients to ascending infections. Clinical signs of acute pyelonephritis may include fever, lethargy, vomiting, anorexia, and renal pain. Laboratory abnormalities may include leukocytosis with a left shift, azotemia, pyuria, bacteriuria, and hematuria. Abdominal radiography may demonstrate renomegaly (Figures 6 and 7), whereas ultrasonographic abnormalities may include renomegaly, hyperechoic renal cortices, renal pelvic dilation, and decreased corticomedullary junction. Animals with chronic pyelonephritis may not have the described clinical signs or abnormalities on diagnostic testing but may still be in renal failure.

*Escherichia coli* is the most common organism isolated in cases of canine bacterial pyelonephritis. Several strains have uropathogenic factors that allow them to colonize the urinary tract, including adhesins that increase bacterial adherence to epithelial cells. Strains of *E. coli*—causing pyelonephritis are more adhesive than those causing bacterial cystitis. Additional virulence factors associated with pyelonephritis-causing *E. coli* include hemolysin, cytotoxic necrotizing factor, aerobactin, and secreted autotransporter toxin.

**Lyme Disease**

Lyme disease is caused by the spirochete bacterium *Borrelia burgdorferi* and is transmitted by ticks of the genus *Ixodes*. Dogs with borreliosis are most frequently asymptomatic, but clinical disease can manifest as lame-
ness, fever, lethargy, and anorexia. Cardiac and neurologic signs are rare. \textsuperscript{41} Dogs typically improve within 48 hours of administration of appropriate antimicrobial therapy. \textsuperscript{41}

Although the incidence of Lyme disease is unknown, the disease has been associated with severe protein-losing glomerulopathy and ARF in dogs. \textsuperscript{41} From 1987 to 1992, several dogs in Lyme-endemic areas were diagnosed with rapidly progressing glomerulopathy. \textsuperscript{42} Labrador retrievers, golden retrievers, and Shetland sheepdogs appear predisposed. \textsuperscript{41} Most affected dogs presented with sudden onset of anorexia, vomiting, lethargy, and weight loss and developed ARF with proteinuria, peripheral edema, and body cavity effusion. Clinical disease progressed rapidly, usually leading to euthanasia or death within 1 to 2 weeks, although three dogs lived for several months. \textsuperscript{41}

Of the dogs tested, all had positive results for \textit{B. burgdorferi} via serology. \textsuperscript{42} Other dogs had spirochetes in renal tissue, and one tested positive for \textit{B. burgdorferi} via urine culture; however, more sensitive molecular techniques found little evidence of \textit{B. burgdorferi} in banked tissue from affected dogs. \textsuperscript{31,41} Further research needs to be conducted to determine whether Lyme disease, vaccination for Lyme disease, or an unidentified organism is associated with the severe renal lesions of the condition called \textit{canine Lyme nephritis}.

\section*{ISCHEMIC CAUSES}

Ischemic causes of ARF include hypovolemia, hypotension, and thromboembolic disease. Disseminated intravascular coagulation due to pancreatitis, immune-mediated disease, neoplasia, heatstroke, or other diseases can lead to ARF (Figure 8). Injury occurs when renal blood flow is reduced because of decreased blood pressure, microthrombi, or renal vasoconstriction. Decreased perfusion leads to accumulation of metabolic wastes, decreased delivery of cellular nutrients, and hypoxia. Stores of ATP are rapidly used, and when ATP deficiency occurs, the ATPase pump is unable to maintain normal cellular transport of sodium, potassium, and calcium. Increased intracellular calcium levels cause further vasoconstriction and cell membrane damage. Cell swelling occurs with accumulation of intracellular sodium. Vasoconstriction and cell swelling lead to vascular stasis and occlusion of the blood supply to the renal cortex, causing further ischemic damage. Decreased delivery of nutrients leads to cell membrane damage and formation of oxygen-derived free radicals, which also damage cell membranes.

\section*{MISCELLANEOUS CAUSES}

Rarely reported causes of ARF in dogs include envenomation by poisonous snakes and bull ants. \textsuperscript{44,45} Snake venom contains nephrotoxins that produce renal ischemia, alterations in renal hemodynamics, and coagulation disorders, all of which can lead to ARF. \textsuperscript{45}

Administration of an iodinated radiographic contrast medium (e.g., sodium iopanoate, diatrizoate meglumine, diatrizoate sodium) has rarely been reported as a cause of ARF in dogs, although it is the third leading cause of
Ureteral obstruction is being increasingly recognized as a cause of ARF in cats. From 1993 to 2003, 37% of 119 cats undergoing dialysis for management of ARF had acute ureteral obstruction. All reported cats with ureteral obstruction presented from 1999 to 2003 and represented 50% of all cases. Most cats presented after sequential obstruction of both ureters due to calcium oxalate ureteroliths (approximately 75% of the reported cases) or organic material. Although most cats presented with severe azotemia (serum creatinine: >7 mg/dl), many appeared less ill than cats with similar degrees of azotemia due to other causes. The physical examination findings included asymmetric and/or painful kidney(s). Radiographic abnormalities included renal asymmetry, bilateral renomegaly, expansion of the retroperitoneal space, or ureteroliths. Ultrasonography diagnosed ureteral obstruction in approximately 75% of cats, although it may take up to 7 days after complete obstruction for ultrasonographic changes to be detected. In 20% to 30% of cats, no discrete mineralized material was identified with routine abdominal imaging. Antegrade pyelography using ultrasonographic guidance to inject contrast medium into the renal pelvis has been used to detect ureteral obstructions that were inapparent with routine radiography or that involved multiple areas of the ureter.

Approximately 25% of cats with ureteral obstruction that were initially managed with hemodialysis had...
spontaneous resolution of the obstruction within 2 to 5 days. An additional 40% with mild uremia were successfully managed with routine medical treatment. In cases in which spontaneous resolution did not occur or hemodialysis was unavailable, surgical resolution was necessary. In the perioperative period, approximately 20% of cats died as a result of surgical complications. In cats surviving the perioperative period, 2-year survival was greater (88%) than in cats managed nonsurgically (66%). Renal transplantation may be an additional treatment option in select cases.

Posturethral obstruction is another potential cause of ARF. Uroliths, mass lesions, foreign bodies, or strictures in the urethral orifice, whether originating from the lower urinary tract or genital tract, can cause urinary outflow obstruction and postrenal ARF.

PROGNOSIS

The mortality rate of ARF varies, depending on the cause, extent of damage, concurrent diseases, and timing and effectiveness of therapy. Mortality associated with all causes of ARF in dogs ranges from 56% to 80%, but include severity of azotemia (initial serum creatinine: >10 mg/dl), hypocalcemia (total serum calcium: <8.6 mg/dl), proteinuria, gender, age, oliguria or anuria, anion gap, and phosphorus concentration. Dogs 7 years of age or older had an approximately ninefold risk of death, whereas oliguric dogs were 20 times more likely to die. The mortality rate also varies by cause: infectious (30%), hemodynamic/metabolic (44%), and toxic (82%). Ethylene glycol–induced ARF carries a poor prognosis, with 88% to 100% of dogs dying or being euthanized.

Less information is available concerning the prognosis in cats with ARF. In one retrospective study of 25 cases, 56% of cats died or were euthanized. Mortality was higher for the 72% of cats that presented with anuria or oliguria; all nonoliguric or polyuric cats survived. All cats that were oliguric and in ARF due to nephrotic causes and those that were anuric died. A second retrospective study assessing 119 cats treated with hemodialysis for ARF showed an overall mortality rate of 48%. Mortality was lowest (25%) in cats that had obstructive ARF.

CONCLUSION

Despite the availability of aggressive medical therapy and dialysis for ARF, the mortality rate remains high. It is important for veterinarians to be aware of new and reemerging causes of ARF in cats and dogs. Recognizing causes of ARF as well as predisposing factors, many of which are preventable and/or hospital acquired, can help decrease the incidence of and mortality due to ARF.

REFERENCES


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**ARTICLE #4 CE TEST**

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1. Which is the most common cause of ARF in dogs and cats?
   - a. ischemia
   - b. nephrotoxicity
   - c. infectious disease
   - d. urinary tract obstruction

2. Which is an approximate overall mortality rate for ARF in dogs?
   - a. 2%
   - b. 15%
   - c. 60%
   - d. 95%

3. Which medication(s) is potentially nephrotoxic?
   - a. penicillin
   - b. ibuprofen
   - c. doxorubicin
   - d. all of the above

4. Which is a reemerging disease that has been confirmed as a cause of ARF in dogs and/or cats?
   - a. FIV
   - b. borreliosis
   - c. leptospirosis

5. Which predispose(s) animals to ARF?
   - a. dehydration
   - b. aminoglycoside
   - c. advanced age
   - d. all of the above

6. Ethylene glycol toxicosis
   - a. can cause neurologic or cardiopulmonary signs.
   - b. has a good prognosis (>80% survival) when patients are managed with hemodialysis.
   - c. is a rare cause of poisoning in dogs.
   - d. leads to the development of severe metabolic alkalosis.
7. Which mechanism has been proven for raisin toxicity in dogs?
   a. hypercalcemia
   b. calcium oxalate formation in renal tubules
   c. heavy metal toxicity
   d. none of the above

8. Which source(s) of cholecalciferol has been associated with ARF in dogs and/or cats?
   a. oversupplemented diets
   b. rodenticides
   c. topical preparations
   d. all of the above

9. Which NSAID is most commonly associated with accidental ingestion in dogs?
   a. ibuprofen
   b. carprofen
   c. meloxicam
   d. aspirin

10. Which organism is most commonly associated with pyelonephritis?
    a. *Pseudomonas* spp
    b. *E. coli*
    c. *Bacteroides* spp
    d. *Staphylococcus* spp