CONTINUING EDUCATION

TREATMENT GUIDELINES FOR CHRONIC KIDNEY DISEASE IN DOGS & CATS:

International Renal Interest Society Recommendations

***Gregory F. Grauer**, DVM, MS, DACVIM*
Kansas State University

Chronic kidney disease (CKD) affects an estimated 1% to 3% of all cats and 0.5% to 1.5% of all dogs.[1] Nephron damage associated with CKD is usually irreversible and can be progressive (**Figure 1**, page 42). CKD is a major cause of morbidity and mortality, especially in older dogs and cats.

Because renal replacement therapy (dialysis and transplantation) is not widely available in veterinary medicine, management of CKD in dogs and cats focuses on:

- Early detection
- Renoprotective treatments designed to slow the progressive loss of nephrons.

INTERNATIONAL RENAL INTEREST SOCIETY

Many different terms have been used to describe renal disease and decreased renal function. Unfortunately, these terms can be confusing due to a lack of standard definition and application (eg, renal insufficiency and end-stage renal disease/failure).

The International Renal Interest Society (IRIS, iris-kidney.com) was created to advance the scientific understanding of kidney disease in small animals and, specifically, to help practitioners better diagnose, understand, and treat canine and feline renal disease.

IRIS has created an internationally recognized set of guidelines for the classification and treatment of kidney disease; these guidelines are available on the IRIS website and address:

- Staging of CKD
- Treatment recommendations for CKD
- Grading of AKI (acute kidney injury).

View the IRIS Guidelines

Staging of CKD
iris-kidney.com/guidelines/staging.html

Treatment Recommendations for CKD
iris-kidney.com/guidelines/recommendations.html

Grading of AKI
iris-kidney.com/guidelines/grading.html

shutterstock.com/DuxX

PEER REVIEWED

STAGING CANINE & FELINE CKD

The IRIS Staging of CKD guidelines were developed as a guide to classifying stable canine and feline CKD in order to both improve communications surrounding CKD and link appropriate diagnostic and therapeutic efforts to patients with varying degrees of CKD.

Learn More

Read **Early Diagnosis of Chronic Kidney Disease in Dogs & Cats: Use of Serum Creatinine & Symmetric Dimethylarginine** in the March/April 2016 issue of *Today's Veterinary Practice*, available at **tvpjournal.com.**

These guidelines have been published in well-known textbooks, such as *Current Veterinary Therapy* and *Textbook of Veterinary Internal Medicine*, and have been adopted by the American and European Societies of Veterinary Nephrology and Urology (asvnu.org and esvnu.eu, respectively).[2,3]

This staging system is not used to make a diagnosis of CKD but is employed following a diagnosis of CKD in order to facilitate appropriate treatment, monitoring, and further diagnostics.

Serum Creatinine Concentration

The IRIS staging system is based primarily on serum creatinine concentrations (**Table 1**) and applies only to dogs and cats that are well hydrated and have stable CKD—stability is documented by < 20% variation in serum creatinine concentrations over at least a 2-week interval.

Note that the lower end of serum creatinine concentrations in Stage 2 lies within the reference interval for many laboratories. Serum creatinine concentration is a relatively insensitive marker of renal function and, therefore, dogs and cats with serum creatinine concentrations near the upper end of the laboratory reference interval may have reduced glomerular filtration rates.

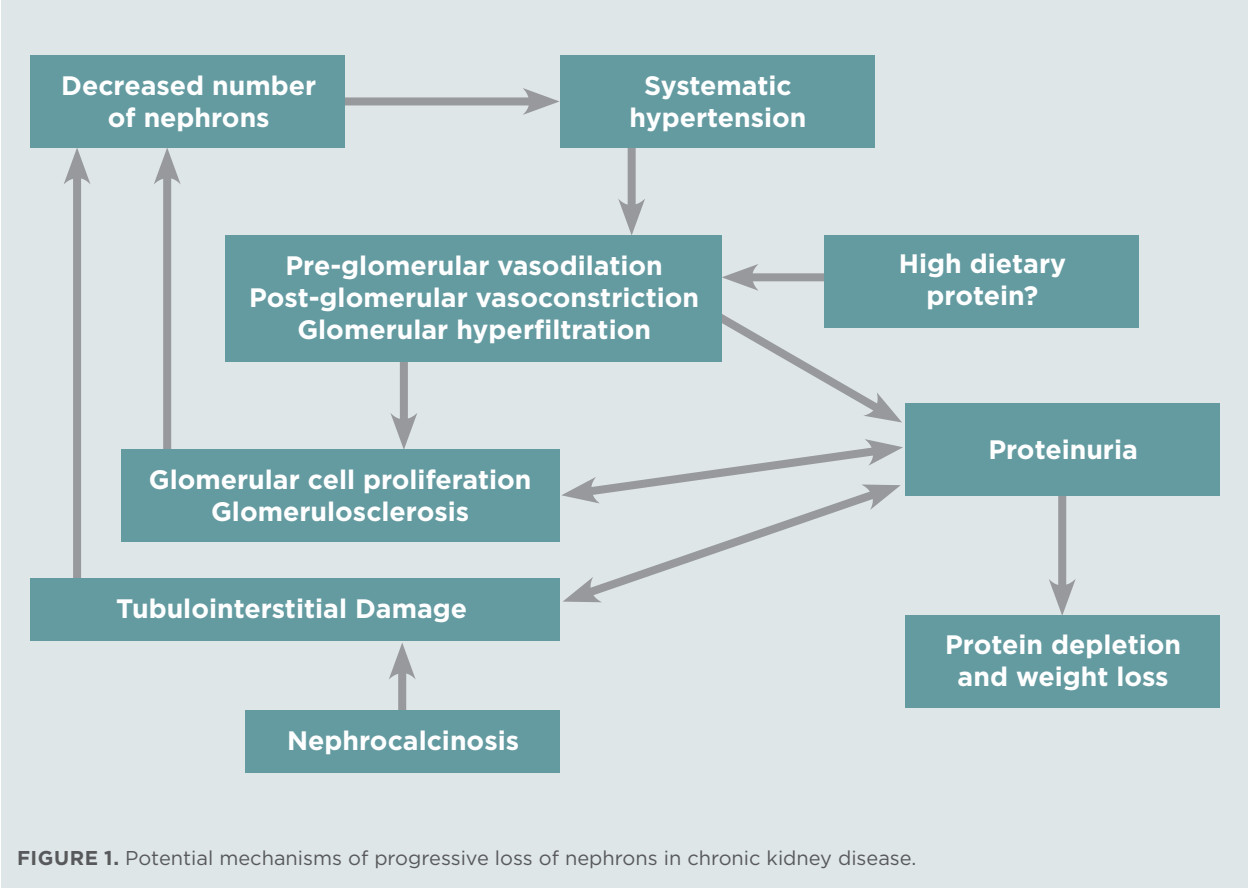

FIGURE 1. Potential mechanisms of progressive loss of nephrons in chronic kidney disease.

TABLE 1 Stages of CKD: Classified by Serum Creatinine Concentration

STAGES OF CKD	SERUM CREATININE CONCENTRATION (MG/DL)	
	CATS	DOGS
Stage 1 Non-azotemic	< 1.6	< 1.4
Stage 2 Non-azotemic to mild renal azotemia	1.6–2.8	1.4–2
Stage 3 Moderate renal azotemia	2.9–5	2.1–5
Stage 4 Severe renal azotemia	> 5	> 5

Serum creatinine concentrations must always be interpreted in light of the patient's muscle mass, urine specific gravity, and physical examination findings in order to rule out pre- and postrenal causes of azotemia.

The IRIS CKD staging system cannot be applied to patients with:

- Pre- or postrenal azotemia
- Acute or decompensated (sometimes termed *acute on chronic*) kidney disease.

The **IRIS stages of CKD** are primarily defined by serum creatinine concentration (**Table 1**) and then further classified by:

- Presence or absence of proteinuria (**Table 2**)
- Systemic hypertension (**Table 3**).

SDMA Concentrations

Interpretation of serum symmetric dimethylarginine (SDMA) concentrations, along with serum creatinine concentrations, may increase the sensitivity for early diagnosis of CKD.[4,5]

Renal Proteinuria

Proteinuria is an important risk factor for the development of azotemia in cats and the progression of azotemia and decreased survival in both dogs and cats.[6-8] Presence or absence of proteinuria is used to substage CKD (**Table 2**) in the IRIS staging system.

TABLE 2 Substages of CKD: Classified by Urine Protein/Creatinine Ratio (UPC)

CLASSIFICATION OF UPC	UPC RATIO	
	CATS	DOGS
Non-proteinuric	< 0.2	< 0.2
Borderline proteinuric	0.2–0.4	0.2–0.5
Proteinuric	> 0.4	> 0.5

Renal proteinuria can be glomerular and/or tubular in origin (ie, excessive filtration, decreased tubular reabsorption, or both). Renal proteinuria is persistent—with at least 2 positive tests separated by 10 to 14 days—and associated with inactive urine sediments.

Urine protein/creatinine ratios (UPCs) > 2 suggest glomerular-range proteinuria, which is rare in cats compared with dogs.

It is important to recognize that the UPC does not differentiate renal proteinuria from proteinuria associated with lower urinary tract inflammation; the clinician needs to make this determination by assessing the patient and urine sediment.

Systolic Blood Pressure

IRIS blood pressure substaging is based, in part, on risk of target organ—eye, brain, heart, and kidney—damage (**Table 3**). In the absence of target organ damage, persistence of hypertension should be documented.

Systolic blood pressure is typically measured by the Doppler methodology in dogs and cats.

TABLE 3 Substages of CKD: Classified by Systemic Blood Pressure & Risk of Target Organ Damage

CLASSIFICATION OF BLOOD PRESSURE	SYSTOLIC BLOOD PRESSURE (MM HG)	RISK OF TARGET ORGAN DAMAGE
Normotensive	< 150	Minimal
Borderline hypertensive	150–159	Low
Hypertensive	160–179	Moderate
Severe hypertension	≥ 180	Severe

PEER REVIEWED

Although it is preferable to measure blood pressure on different days, it is acceptable to perform 2 measurements at least 2 hours apart. Most clinicians consider systolic hypertension to be > 160 mm Hg and initiate treatment at that point.

DIAGNOSTIC APPROACH AFTER STAGING

In general, the diagnostic approach to a patient once CKD has been identified and staged focuses on 3 areas (**Table 4**).

1. **Characterization of primary renal disease and/or complicating disease processes.**
   Further definition of the renal disease—beyond a standard minimum database—should include diagnostics to rule out potentially treatable conditions/complications; for example, urine culture for urinary tract infection and kidney imaging for renal lymphosarcoma.

2. **Characterization of the stability of renal disease and function.**
   Stability of renal function should be assessed by serial monitoring of abnormalities identified during initial characterization of the renal disease (**Table 5**).

   If the same laboratory and methodology are used, **increases in serum creatinine > 0.3 mg/dL are suggestive of changes in renal function** rather than assay variability. It is important to rule out dehydration as a cause of increasing serum creatinine concentrations.

3. **Characterization of patient problems associated with decreased renal function.**
   Patient problems associated with decreased renal function (**Table 4**) may include anorexia, nausea, vomiting, weight loss, dehydration, acidosis, potassium depletion, and anemia.

TABLE 4 Diagnostic & Treatment Considerations Linked to IRIS CKD Stages

OPTIMUM IRIS STAGES	DIAGNOSTIC & TREATMENT FOCUS	CONSIDERATIONS	
		POTENTIAL PROBLEMS	DIAGNOSTICS/TREATMENT
Stage 1 **Stage 2** **Early Stage 3**	**Assess primary disease and complicating disorders** Monitoring at least Q 6 months	Renal infiltrative disease Renal lymphosarcoma	Radiographs, ultrasound ± FNA, chemotherapy
		Obstructive uropathy Ureteral obstruction	Radiographs, ultrasound ± FNA, chemotherapy, SC ureteral bypass
		Hypercalcemic nephropathy	Serum Ca and iCa assessment, NaCl fluid therapy, furosemide diuresis
Stage 2 **Stage 3** **Early Stage 4**	**Assess CKD stability or progression** Monitoring at least Q 3 months	Nephrocalcinosis	Renal diets, intestinal phosphorus binders
		Hypertension	CCAs, ACE inhibitors, ARBs
		Proteinuria	ACE inhibitors, CCAs, ARBs
Late Stage 3 **Stage 4**	**Assess patient problems** Monitoring at least Q 1–2 months	Anorexia, nausea, vomiting	Appetite stimulants, antiemetics, H2 receptor blocker, proton pump blockers
		Metabolic acidosis	Dietary alkalization
		Potassium depletion	Potassium supplementation
		Dehydration	Fluid therapy
		Anemia	Recombinant erythropoietin
		Calorie malnutrition	Appetite stimulants, dietary variety, feeding tube placement
		Uremia	Enteric dialysis

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; Ca = calcium; CCA = calcium channel antagonist; CKD = chronic kidney disease; FNA = fine-needle aspiration; iCa = ionized calcium; NaCl = sodium chloride

Timing of Diagnostics

In the **early stages of CKD**, characterization of primary renal disease and/or complicating disease processes, as well as determining disease stability, are most important—when appropriate treatment has the greatest potential to improve or stabilize renal function.

In the **later stages of CKD**, characterization of patient problems becomes more important—when clinical signs tend to be more severe. In these later stages, diagnostic and subsequent therapeutic efforts should be directed at patient problems.

THERAPEUTIC APPROACH TO CKD

Therapy Tailored to CKD Stage

Similar to the diagnostic approach, the therapeutic approach to CKD should be tailored to each individual patient and that patient's stage of disease (**Tables 4 and 6**). Serial monitoring of the patient—after treatment has been initiated—allows the clinician to modify treatment based on patient response (**Table 5**).

TABLE 5 Serial Monitoring of Patients with CKD

FREQUENCY OF MONITORING		MONITORING SHOULD INCLUDE:
Stage 1 **Stage 2** **Early Stage 3**	At least Q 6 months	• Serum biochemistry profile • Urinalysis • Quantitation of proteinuria • Blood pressure measurement • ± urine culture and ultrasound examination
Stage 2 **Stage 3** **Early Stage 4**	At least Q 3 months	
Late Stage 3 **Stage 4**	At least Q 1–2 months	

TABLE 6 Therapeutic Approach Based on IRIS Stage of CKD

	STAGE 1 CKD	STAGE 2 CKD	STAGE 3 CKD	STAGE 4 CKD
Pursue additional diagnostics (eg, urine C/S, urinary tract imaging)	✓	✓	✓	✓
Discontinue all potentially nephrotoxic drugs	✓	✓	✓	✓
IDENTIFY AND CORRECT/TREAT:				
Prerenal or postrenal disorders	✓	✓	✓	✓
Primary disease processes or complicating disorders	✓	✓	✓	
Hypertension and renal proteinuria	✓	✓	✓	✓
CONSIDER RENOPROTECTIVE TREATMENTS:				
Renal diet		✓	✓	✓
ACE inhibitor, CCA, ARB	✓	✓	✓	✓
Calcitriol[a]		✓	✓	✓
Reduce phosphorus intake (eg, renal diet, enteric phosphate binders)		✓	✓	✓
Monitor for metabolic acidosis[b]		✓	✓	✓
Symptomatic treatment to improve quality of life			✓	✓
Monitor serum creatinine[c]	✓	✓	✓	✓
Address anorexia and calorie malnutrition[d]			✓	✓
Assess CKD stability/progression	Q 6 months	Q 3–6 months	Q 1–3 months	Q 1–2 months

a. Avoid calcium containing enteric phosphate binders or monitor closely for hypercalcemia.
b. Consider adding oral sodium bicarbonate or potassium bicarbonate to renal dietary therapy.
c. In patients receiving vasoactive drugs for hypertension and/or proteinuria.
d. Consider appetite stimulants, antiemetics, and gastric acid blockings drugs, but correction of metabolic deficits/excesses is more important.
ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blockers; CCA = calcium channel antagonist; C/S = culture and sensitivity

PEER REVIEWED

For example, disease specific treatments for ureteroliths and bacterial pyelonephritis, as well as treatments designed to slow the progression of renal disease (renoprotective treatments), are of most value in the earlier stages of CKD. In the later stages of CKD, treatment tends to be focused on quality of life and managing clinical signs associated with decreased renal function.

Learn More

Read **Nutritional Management of Chronic CKD in Dogs & Cats** (March/April 2016), which details the efficacy of therapeutic diets, key nutrients in CKD, and role of body condition, available at **tvpjournal.com**. This article is RACE-approved for CE credit.

Renoprotective Treatments

Renoprotective treatments include:

- Dietary change designed to reduce serum phosphorus concentrations and decrease soft tissue mineralization[9,10]
- Potentially angiotensin-converting enzyme (ACE) inhibitors, calcium channel antagonists (CCA), and angiotensin receptor blockers (ARB) to normalize systemic and intraglomerular blood pressures and reduce proteinuria (**Table 7**)[11-14]
- Potentially, calcitriol supplementation.

CCAs are typically the first line of defense for moderate to severe hypertension in cats, while ACE inhibtors are typically the first line of defense for hypertension in dogs as well as proteinuria in dogs and cats.

There is some evidence in dogs and cats that CCAs upregulate the renin–angiotensin–aldosterone system and, therefore, combined treatment with an ACE inhibitor is often recommended.[15,16] Vasoactive drugs (ACE inhibitors, CCAs, and ARBs) should not be administered to dehydrated patients.

In patients in which hypertension and/or proteinuria are refractory to initial ACE inhibitor treatment, the standard dose can be doubled at least once, combined with a CCA, or the ACE inhibitor may be replaced with an ARB (eg, telmisartan).

For patients with Stage 2 CKD, calcitriol supplementation (0.5–1 ng/kg PO, separate from feeding) is a potentially renoprotective treatment in dogs[17] but unproven in cats. Patients should be normocalcemic, with serum phosphorus concentrations within the target range (see **Treatment Goals**), prior to calcitriol supplementation.

Phosphorus Reduction

Reduction of phosphorus intake is a major treatment goal for dogs and cats with Stage 2 and beyond CKD. The first line of defense against higher serum phosphorus concentrations is a gradual transition to a renal diet. A gradual transition over several weeks from a maintenance diet to a renal diet helps avoid any aversion to the renal diet.

TABLE 7 Dosages of Drugs for Management of CKD

DRUG CLASSIFICATION	DRUG	DOSAGE
ACE inhibitor	Benazepril	0.5–1 mg/kg PO Q 24 H
	Enalapril	0.5–1 mg/kg PO Q 24 H
Angiotensin receptor blocker	Telmisartan	Cats: 1 mg/kg PO Q 24 H
Antidepressant[a]	Mirtazapine	1.5–2 mg/cat PO Q 24 H
Antiemetic	Maropitant	1 mg/kg PO or SC Q 24 H
Calcium channel antagonist	Amlodipine	Dogs: 0.0625–0.25 mg/kg PO Q 24 H Cats: 0.625–1.25 mg/cat PO Q 24 H
Proton pump inhibitor	Omeprazole	Dogs: 0.7 mg/kg PO Q 24 H Cats: 1 mg/kg PO Q 12 H

a. Used for appetite stimulation in cats

Most renal diets are not only phosphorus restricted but:

- Contain reduced amounts of protein and salt
- Are supplemented with omega-3 fatty acids
- Are alkalinized to help offset the metabolic acidosis associated with CKD.

Feline renal diets are also often supplemented with potassium.

Enteric phosphate binders are the second line of defense if serum phosphorus is > 4.6 mg/dL after dietary phosphorus restriction (see **Treatment Goals**). Many different enteric phosphate binders exist but all need to be well mixed with the diet or administered at the time of feeding.

To increase efficacy, the binder should be in the gut when phosphorus from the diet is also there. The dose of the binder required to meet the target serum phosphorus goal will vary with the amount of phosphorus being fed and the stage of CKD.

Use of calcium containing enteric phosphate binders in dogs and cats receiving calcitriol should be avoided or monitored closely for hypercalcemia (total and ionized calcium concentrations). CKD dogs with a product of serum calcium × phosphorus concentrations > 70 mg/dL have reduced survival times.[18]

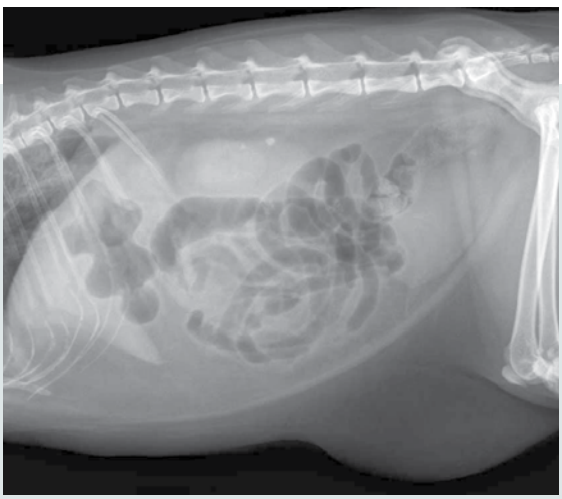

FIGURE 2. Lateral abdominal radiograph of a cat with uroliths.

Treatment Goals

Goals for antihypertensive treatment and antiproteinuric treatment are a systolic blood pressure < 160 mm Hg and a UPC < 0.4 in cats and 0.5 in dogs. Alternatively, a reduction of > 50% of the baseline UPC is an acceptable response.

Target serum phosphorus concentrations are:

- Stage 2 CKD: > 2.7 but < 4.6 mg/dL
- Stage 3 CKD: > 2.7 but < 5 mg/dL
- Stage 4 CKD: > 2.7 but < 6 mg/dL

Note that most laboratory reference intervals for serum phosphorus include concentrations much higher than 4.6 mg/dL.

IRIS TREATMENT RECOMMENDATIONS

Stage 1 CKD Patients

1. **Identify and correct any prerenal or postrenal disorders.** Dehydration is the most common prerenal abnormality encountered, especially if urine-concentrating ability is compromised. Any clinical or suspected subclinical dehydration should be corrected with isotonic, polyionic replacement fluid solutions, such as lactated Ringer's solution either IV or SC.

2. **Identify and treat any treatable primary disease processes** (eg, renal lymphoma and hypercalcemia) or complicating disorders (eg, urinary tract infections and ureteroliths).

3. **Pursue additional diagnostics recommended for Stage 1 CKD patients,** including:

- Urine culture and sensitivity: Many urinary tract infections are subclinical in CKD patients
- Urinary tract radiography: Ideal for localization of radiopaque uroliths (**Figure 2**)
- Urinary tract ultrasound: Ideal for assessing kidney tissue architecture and renal pelvic dilation.

4. **Identify and treat hypertension and renal proteinuria.** In Stage 1 CKD patients, a systolic blood pressure > 160 mm Hg is indicative of hypertension, while a UPC > 0.4 in cats and

> 0.5 in dogs indicates renal proteinuria. Dietary sodium and protein reduction (eg, a renal diet) combined with ACE inhibitors, CCAs, and ARBs are used to reduce hypertension and proteinuria.

5. **Discontinue all potentially nephrotoxic drugs.**

6. **Assess CKD stability or progression by monitoring patients at least twice a year.** Dogs and cats with Stage 1 CKD (**Table 8**) are at risk for kidney disease progression; however, not all Stage 1 CKD patients progress to become azotemic. Those with borderline hypertension and proteinuria should be monitored closely.

Stage 2 CKD Patients

1. **In both dogs and cats, pursue all treatments for Stage 1.**

2. **Identify and treat any primary renal disease or complicating condition**, which is still an important goal in Stage 2 CKD. Dogs and cats with mid to late Stage 2 CKD often have progressive loss of renal function, although the rate of renal disease progression can be variable.

3. **Reduce phosphorus intake with renal diets and enteric phosphate binders** (if needed to meet goals).—This is a major treatment goal for dogs and cats with Stage 2 and beyond CKD.

4. **Consider calcitriol supplementation**—a potentially renoprotective treatment in dogs and cats. In dogs and cats receiving calcitriol, avoid use of calcium containing enteric phosphate binders or monitor patients closely for hypercalcemia.

5. **Monitor patients for metabolic acidosis.** Stage 2 CKD patients should be monitored for metabolic acidosis by measuring serum bicarbonate or total CO_2 concentrations. If necessary, renal dietary therapy may be supplemented with oral sodium bicarbonate or potassium bicarbonate in order to maintain serum bicarbonate concentrations in the 18 to 24 mmol/L range.

6. **Assess CKD stability or progression by monitoring patients Q 3 to 6 months.**

Stage 3 CKD Patients

1. **In both dogs and cats, pursue all treatments for Stage 1 and 2 CKD.**

2. **Continue renoprotective treatments** (eg, renal diets, antihypertensive and antiproteinuric treatments) as Stage 3 CKD patients have progressive renal disease and it is important—as in State 2 CKD patients—to slow disease progression.

TABLE 8 Abnormalities Compatible with Stage 1 Non-azotemic CKD

Kidney palpation or imaging abnormalities
Decreased urine concentrating ability without an identifiable nonrenal cause
Persistent proteinuria of renal origin
Increases in serum creatinine concentration (> 0.3 mg/dL over time) in which prerenal influences, such as dehydration and increases in muscle mass, have been ruled out[a]
Persistent increases in SDMA (> 14 mcg/dL) with a serum creatinine concentration < 1.4 mg/dL in dogs or < 1.6 mg/dL in cats
Kidney biopsy results (**Figure 3**)[b]

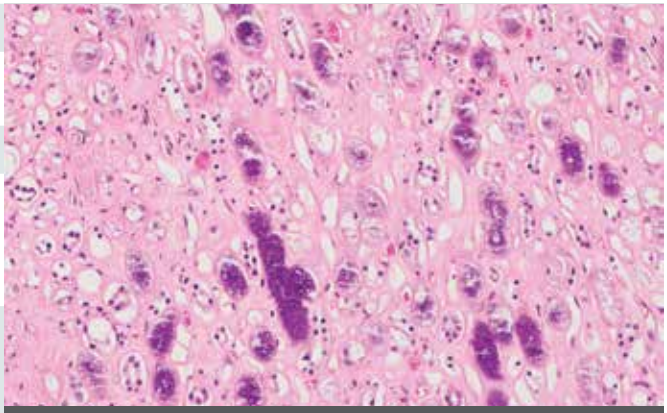

FIGURE 3. Histopathology of CKD showing tubular mineralization (purple).

NOTES
a. Increases in serum creatinine concentration can occur within the laboratory reference interval (eg, an increase in serum creatinine concentration from 0.6 to 1.2 mg/dL over several years in a dog or cat can be associated with > 50% nephron loss).
b. Kidney biopsy is not routinely recommended in CKD.

PEER REVIEWED

Gregory F. Grauer
Gregory F. Grauer, DVM, MS, DACVIM (SAIM), is a professor and Jarvis Chair of Medicine, Department of Clinical Sciences, at Kansas State University College of Veterinary Medicine. His clinical and research interests involve the small animal urinary system. He is on the board of directors of the IRIS and American Society of Veterinary Nephrology and Urology. Dr. Grauer received his postgraduate training in internal medicine at Colorado State University. He has been a faculty member at University of Wisconsin and Colorado State University Colleges of Veterinary Medicine.

3. **Initiate symptomatic treatment to improve quality of life** (**Table 4**) because many dogs and cats with Stage 3 CKD, especially late Stage 3 CKD, begin showing clinical signs.

4. **Assess CKD stability or progression by monitoring patients Q 3 months**; those with early Stage 3 CKD can be monitored Q 6 months, while those with late Stage 3 CKD should be monitored Q 1 to 2 months.

Stage 4 CKD Patients

1. **In both dogs and cats, pursue all treatments for Stage 1, 2, and 3 CKD.**

2. **Continue renoprotective treatments (eg, renal diets, antihypertensive and antiproteinuric treatments)** as these treatments are still important in early Stage 4 CKD patients but invariably the management focus shifts to making the patient as comfortable as possible given its renal failure.

3. **Continue symptomatic treatment to improve quality of life** (**Table 4**). Owners frequently—and rightfully—equate nausea, decreased appetite, vomiting, and weight loss with poor quality of life.

4. **Stop the catabolic spiral of calorie malnutrition**—one of the primary management goals in Stage 4 CKD (**Table 4**). Appetite stimulants, antiemetics, and gastric acid blocking drugs become important in these patients (**Table 7**), but correction of metabolic deficits (eg, dehydration) and excesses (eg, hyperphosphatemia) is a higher priority.[19,20]

5. **Monitor serum creatinine concentrations closely in Stage 4 CKD dogs and cats** that are being treated with vasoactive drugs for hypertension and/or proteinuria.

6. **Reevaluate patients Q 1 to 2 months.**

IN SUMMARY

Understanding the diagnostic and therapeutic priorities based on the stage of CKD facilitates appropriate management of dogs and cats with CKD. Identification and correction of any primary or complicating diseases are most important in Stage 1 and 2 patients. Renoprotective treatments are most important in Stage 2 and 3 patients. Symptomatic patient therapy to improve quality of life is most important in Stage 4 patients. **TVP**

References

1. Brown SA. Management of chronic kidney disease. In Elliott J, Grauer GF (eds): *BSAVA Manual of Canine and Feline Nephrology and Urology*, 2nd ed. Gloucester (UK): BSAVA, 2007, pp 223-230.
2. Bonagura JD, Twedt DC. *Kirk's Current Veterinary Therapy XV*. St. Louis: Elsevier, 2014.
3. Ettinger SJ, Feldman EC. *Textbook of Veterinary Internal Medicine Expert Consult*, 7th ed. St. Louis: Elsevier, 2010.
4. Hall JA, Yerramilli M, Obare E, et al. Comparison of serum concentrations of symmetric dimethylarginine and creatinine as kidney function biomarkers in cats with chronic kidney disease. *J Vet Intern Med* 2014; 28(6):1676-1683.
5. Hall JA, Yerramilli M, Obare E, et al. Serum concentrations of symmetric dimethylarginine and creatinine in dogs with naturally occurring chronic kidney disease. *J Vet Intern Med* 2016; 30(3):794-802.
6. Jepson RE, Brodbelt D, Vallance C, et al. Evaluation of predictors of the development of azotemia in cats. *J Vet Intern Med* 2009; 23(4):806-813.
7. Jacob F, Polzin DJ, Osborne CA, et al. Evaluation of the association between initial proteinuria and morbidity rate or death in dogs with naturally occurring chronic renal failure. *JAVMA* 2005; 206(3):393-400.

References continued on page 53

Glossary

ACE angiotensin-converting enzyme
ARB angiotensin receptor blocker
CCA calcium channel antagonist
CKD chronic kidney disease
IRIS International Renal Interest Society
SDMA serum symmetric dimethyl arginine
UPC urine protein/creatitine ratio

PEER REVIEWED

Treatment Guidelines for Chronic Kidney Disease in Dogs & Cats

LEARNING OBJECTIVES

After reading this article, participants will be able to identify the IRIS stages of CKD and understand how staging guides further diagnostics and treatment.

OVERVIEW

This article illustrates appropriate diagnostic and therapeutic management of dogs and cats with CKD based on differentiation of the patient's stage of CKD.

This article is **RACE-approved for 1 hour of continuing education credit**. To receive credit, take the approved test online at vetmedteam.com/tvp.aspx **(CE fee of $5/article)**.

1. **In Stage 1 and early stage 2 CKD, the primary treatment strategy is:**
   a. Reducing patient clinical signs and improving quality of life
   b. Renoprotective therapies to slow progression of kidney disease
   c. Identification and correction of primary or complicating diseases
   d. Correction of anemia
   e. Correction of acidosis

2. **In mid Stage 2 and Stage 3 CKD, the primary treatment strategy is:**
   a. Reducing patient clinical signs and improving quality of life
   b. Renoprotective therapies to slow progression of kidney disease
   c. Identification and correction of primary or complicating diseases
   d. Correction of anorexia/calorie malnutrition
   e. Correction of potassium depletion

3. **In late Stage 3 and Stage 4 CKD, the primary treatment strategy is:**
   a. Reducing patient clinical signs and improving quality of life
   b. Renoprotective therapies to slow progression of kidney disease
   c. Identification and correction of primary or complicating diseases
   d. Reduction of serum SDMA concentrations
   e. Gradual transition to a renal diet

4. **Serum creatinine concentrations can be influenced by:**
   a. Prerenal dehydration
   b. Postrenal urethral obstruction
   c. Patient muscle mass
   d. Breed characteristics
   e. All of the above

5. **Which of the following is not compatible with a diagnosis of Stage 1 CKD?**
   a. Primary polydipsia
   b. Renal palpation or imaging abnormalities
   c. Persistent renal proteinuria
   d. Persistent increases in SDMA in a non-azotemic patient
   e. Increase in serum creatinine > 0.3 mg/dL within the reference interval that is not associated with changes in muscle mass or hydration

6. **The IRIS CKD staging system should be used as a guide to diagnose CKD in dogs and cats.**
   a. True
   b. False

7. **Renal proteinuria is:**
   a. Always associated with a UPC > 1
   b. Intermittent and often transient
   c. Frequently associated with an active urine sediment
   d. Either of glomerular or tubular origin
   e. Of no prognostic significance in CKD

8. **Which of the following is not thought to contribute to renal disease progression?**
   a. Soft tissue mineralization
   b. Intraglomerular hypertension
   c. Proteinuria
   d. Anorexia/calorie malnutrition
   e. Glomerulosclerosis

9. **Dogs and cats with Stage 1 CKD will invariably progress to Stage 2 CKD.**
   a. True
   b. False

10. **Which of the following is most important in the management of anorexia/calorie malnutrition?**
    a. Correction of dehydration
    b. Use of appetite stimulants
    c. Use of gastric acid blocking drugs
    d. Use of antiemetics
    e. Providing dietary variety

NOTE

Questions online may differ from those here; answers are available once CE test is taken at **vetmedteam.com/tvp.aspx**. Tests are valid for 2 years from date of approval.

CHRONIC KIDNEY DISEASE IN DOGS & CATS continued from page 50

8. Syme HM, Markwell PJ, Pfeiffer D, Elliott J. Survival of cats with naturally occurring chronic renal failure is related to severity of proteinuria. *J Vet Intern Med* 2006; 20(3):528-535.
9. Polzin DJ, Osborne CA, Ross S, Jacob F. Dietary management of feline chronic renal failure: Where are we now? In what direction are we headed? *J Feline Med Surg* 2000; 2(2):75-82.
10. Jacob F, Polzin DJ, Osborne CA, et al. Clinical evaluation of dietary modification for treatment of spontaneous renal failure in dogs. *JAVMA* 2002; 220(8):1163-1170.
11. Grauer GF, Greco DS, Getzy DM, et al. Effects of enalapril versus placebo as a treatment for canine idiopathic glomerulonephritis. *J Vet Intern Med* 2000; 14(5):526-533.
12. King JN, Gunn-Moore DA, Tasker S, et al. Tolerability and efficacy of benazepril in cats with chronic kidney disease. *J Vet Intern Med* 2006; 20(5):1054-1064.
13. Jenkins TL, Coleman AE, Schmiedt CW, Brown SA. Attenuation of the pressor response to exogenous angiotensin by angiotensin receptor blockers and benazepril hydrochloride in clinically normal cats. *Am J Vet Res* 2015; 76(9):807-813.
14. Sent U, Gossl R, Elliott J, et al. Comparison of efficacy of long-term oral treatment with telmisartan and benazepril in cats with chronic kidney disease. *J Vet Intern Med* 2015; 29:1479-1487.
15. Atkins CE, Rausch WP, Gardner SY, et al. The effect of amlodipine and the combination of amlodipine and enalapril on the renin-angiotensin-aldosterone system in the dog. *J Vet Pharmacol Ther* 2007; 30(5):394-400.
16. Jepsen RE, Syme HM, Elliott J. Plasma renin activity and aldosterone concentrations in hypertensive cats with and without azotemia and in response to amlodipine besylate. *J Vet Intern Med* 2014; 28(1):144-153.
17. Polzin D, Ross S, Osborne C, et al. Clinical benefit of calcitriol in canine chronic kidney disease (abstract). *J Vet Intern Med* 2005; 19:433.
18. Lippi I, Guidi G, Marchetti V, et al. Prognostic role of the product of serum calcium and phosphorus concentrations in dogs with chronic kidney disease: 31 cases (2008-2010). *JAVMA* 2014; 245(10):1135-1140.
19. Parkinson S, Tolbert K, Messenger K, et al. Evaluation of the effect of orally administered acid suppressants on intragastic pH in cats. *J Vet Intern Med* 2015; 29(1):104-112.
20. Quimby JM, Brock WT, Moses K, et al. Chronic use of maropitant for the management of vomiting and inappetence in cats with chronic kidney disease: A blinded, placebo-controlled clinical trial. *J Fel Med Surg* 2015; 17(8):692-697.