

CANINE CHRONIC KIDNEY DISEASE

Current Diagnostics & Goals for Long-Term Management

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Chronic kidney disease (CKD) is an irreversible and progressive deterioration of renal function, resulting from a decreased number of functional nephrons. Unfortunately, the compensatory mechanisms that respond to nephron loss (glomerular hypertension, hyperfiltration) help facilitate progression of CKD, potentially contributing to it more so than the original injury (**Table 1**).

Patients of any age may develop CKD, but the greatest incidence is in geriatric patients. However, congenital renal diseases, including dysplasia and various glomerulopathies, may produce CKD at very early ages. Once diagnosed, CKD typically remains a life-long condition.

Table 1. Causes of CKD

- Calculi/obstruction
- Familial renal disease
- Infection
- Inflammation
- Ischemia
- Unknown
- Vascular injury

PRESENTATION

Medical History

A thorough medical history plays 2 essential roles; it:

1. Helps determine a management plan by assessing severity of polyuria and polydipsia, diet, appetite, change in body mass, and energy level.
2. Provides a baseline—the degree of illness related to CKD at presentation—to use for comparison after therapeutic interventions have been implemented.

Clinical Signs

Early signs of CKD may be mild, even inapparent to the pet owner. Because isosthenuria and azotemia do not develop until 66% and 75% nephron loss, respectively, most renal function has been lost by onset of clinical signs.

Common clinical signs include:

- Polyuria and compensatory polydipsia
- Decreasing appetite, weight loss, and lethargy
- Gastrointestinal (GI) signs, which may be present in early CKD, but are very common in moderate to advanced CKD.

Physical Examination

During a physical examination in patients with suspected or confirmed CKD, pay particular attention to body condition scoring, cardiovascular status, evidence of dehydration, and renal palpation.

- **Muscle wasting** may indicate poor nutritional status.

- **New heart murmurs** may indicate a physiologic flow murmur due to anemia or hypertension (however, sick or febrile patients should also be evaluated for endocarditis).
- **Dehydration** is common in CKD, typically resulting from patients' inability to ingest enough water to compensate for increased urinary losses.
- **Assessment of renal size and shape**, and for the presence of pain, should always be performed, but may be difficult in medium or large breed dogs.

Specific system examinations include:

- **Fundic** examination to assess for vessel tortuosity and retinal detachment, which may suggest systemic hypertension
- **Rectal** examination to evaluate for evidence of melena or hematochezia, which may indicate uremic ulcers.

DIAGNOSTICS

A thorough diagnostic evaluation (**Table 2**) can confirm the diagnosis of CKD. These tests may identify underlying causes, ongoing renal injury, and consequences of CKD, providing information about prognosis and treatment goals.

Azotemia Interpretation

- **Persistent azotemia** (despite normal hydration status) can confirm CKD. However, since 75% nephron loss occurs before azotemia, this criteria only identifies the most advanced cases.
- **Isosthenuria** may be seen earlier (66% nephron loss); however, without azotemia, all other causes of isosthenuria need to be excluded before attributing it to CKD.
- **Extrarenal factors** may alter creatinine or blood urea nitrogen (BUN) when interpreting azotemia:
 - » Creatinine: Decreased in patients with muscle wasting
 - » BUN: Increased in patients with GI bleeding or those consuming a high-protein diet; decreased with malnutrition, severe protein restriction, or synthetic liver failure
- **Prerenal or postrenal factors** may concurrently contribute to azotemia:
 - » Prerenal factors: Consider decreased renal perfusion, most commonly seen in dehydrated, hypovolemic, or hypotensive patients
 - » Postrenal factors: Consider unilateral ureteral obstruction, which can be ruled out by abdominal radiographs and ultrasound; may also manifest with renal pain and abnormal renal palpation.

ACUTE VERSUS CHRONIC RENAL DISEASE

History, physical examination, and diagnostic findings often indicate whether a patient has acute or chronic renal disease (**Table**). With acute-on-chronic renal disease, new injury occurs in patients with background renal insufficiency. Findings suggestive of CKD include:

- **History:** In preceding weeks to months, polyuria/polydipsia, weight loss, and decreased appetite
- **Physical examination:** Small, irregular kidneys that are nonpainful
- **Diagnostics:** Degree of azotemia more severe than clinical signs; normocytic, normochromic nonregenerative anemia typical

TABLE. Comparison of Acute and Chronic Renal Disease

	ACUTE	CHRONIC
Degree of illness	Clinical signs more severe than azotemia	Azotemia more severe than clinical signs
History	Lethargy, vomiting	Decreased appetite, PU/PD, weight loss
Kidney size/shape	Normal/large ± pain	Small, irregular, BKLK
Packed cell volume	Normal/regenerative/acute hemorrhage	Normocytic/normochromic/nonregenerative anemia
Potassium	Normal to high	Cats: Typically low Dogs: Mild hyperkalemia common

BKLK=big-kidney-little-kidney; PD=polydipsia; PU=polyuria

Glomerular Filtration Rate

Glomerular filtration rate (GFR) is the gold standard measurement of renal function; however, its measurement is rarely indicated in patients with CKD. Creatinine and, to a lesser extent, BUN are correlated with GFR, but, as noted earlier, GFR must be reduced by 75% before azotemia is seen. However, measurement of GFR (typically through iohexol or creatinine clearance testing) may confirm reduced renal function in isosthenuric patients.

IRIS STAGING SYSTEM

A tiered stratification system has been proposed by the International Renal Interest Society (IRIS) to help provide guidelines for clinical management of CKD. Staging is based on serum creatinine values, with substages identified for blood pressure and proteinuria (**Table 3**).

Importance of Hydration

Treatment goals and recommendations are specific to IRIS CKD stage. Since prerenal contributions will often increase the degree of azotemia to the next stage, normal renal perfusion (adequate patient hydration and effective circulating volume) should be restored before determining the patient's stage of CKD.

TABLE 2. CKD Diagnostics

Blood pressure measurement
Complete blood count, with reticulocyte count
Serum biochemical profile, with electrolytes and acid-base measurement
Urinalysis + urine protein:creatinine ratio
Urine culture and susceptibility
Urine system imaging (ultrasound and/or radiographs)

TABLE 3. IRIS CKD Staging System

	Dog	Cat
SERUM CREATININE (MG/DL)		
Stage 1	< 1.4	< 1.6
Stage 2	1.4–2	1.6–2.8
Stage 3	2.1–5	2–5
Stage 4	> 5	> 5
PROTEINURIA SUBSTAGING (based on UPC ratio)		
Proteinuric (P)	> 0.5	> 0.4
Borderline Proteinuric (BP)	0.2–0.5	0.2–0.4
Nonproteinuric (NP)	< 0.2	< 0.2
BLOOD PRESSURE SUBSTAGING		
	Systolic Arterial Pressure (AP) (mm Hg)	Diastolic Arterial Pressure (mm Hg)
AP Stage 0	< 150	< 95
AP Stage I	150–160	95–99
AP Stage II	160–179	100–119
AP Stage III	≥ 180	≥ 120

AP = arterial pressure; BP = borderline proteinuric; NP = nonproteinuric; P = proteinuric; UPC = urine protein:creatinine

Stage Determination

Including the IRIS CKD stage in the medical record relays important information about the severity of CKD. For example, if a dog has a creatinine of 2.5 mg/dL, urine protein:creatinine (UPC) ratio of 1, and arterial blood pressure of 155 mm Hg, its IRIS CKD stage would be considered **IRIS 3 P AP 1**, or:

- Serum creatinine: **IRIS 3** (Stage 3)
- Proteinuria substaging: **P** (Proteinuric)
- Blood pressure substaging: **AP I** (Arterial Pressure Stage 1).

TREATMENT GOALS

Treatment of CKD should be individually tailored to each patient. Although not all interventions have been evaluated by clinical trials, some evidence-based information supports their role in management of CKD. IRIS CKD stage management guidelines are listed in **Table 4**; medications to help achieve treatment goals are listed in **Table 5**.

Uremia

As stated earlier, IRIS CKD staging should be applied to patients only after exclusion of pre- and postrenal contributions.

Uremic toxins, many of which are byproducts of protein metabolism, are solutes that accumulate due to decreased renal clearance, causing detrimental effects. Urea and creatinine are not significant uremic toxins; however, they serve as surrogate markers, providing some information on renal function and degree of uremic toxin retention.

Hydration

As CKD is irreversible, decreased GFR caused by intrinsic renal dysfunction cannot be improved. Hypovolemic or dehydrated patients will have decreased renal perfusion, causing prerenal reduction in GFR, which is complicated if patients cannot voluntarily maintain hydration.

Measures should be taken to prophylactically maintain hydration in patients that cannot do so on their own (urine output exceeds fluid intake).

→THERAPEUTIC GOAL: Maintain Hydration

- Feed canned food diets; many patients will tolerate additional water added to canned food.
- Offer low- or no-sodium chicken broth.
- Feeding tubes (esophagostomy, gastric) can provide access for water, medication, and nutrition delivery.
- Subcutaneous fluids can be helpful, but contain large amounts of sodium, which some CKD patients may not tolerate, contributing to hypertension.
- Consider feeding prescription renal diet (see **Nutritional Therapy**).

GI Complications

Antacids and antiemetics are useful for managing GI complications of uremia. Due to diminished ability to produce erythropoietin, dogs with CKD take longer to normalize anemia related to GI ulcers.

→THERAPEUTIC GOAL: Manage GI Complications of Uremia

- **Proton pump inhibitors:** More effective than histamine antagonists for neutralizing gastric acid secretion; no dose adjustment is required in patients with CKD

TABLE 4. IRIS CKD Stage Management Guidelines

	Goal	First-line Therapy	Additional Therapy
IRIS CKD STAGE 1			
Phosphorus	< 4.5 mg/dL	Renal diet	Phosphate binders
UPC	< 2	Renal diet	ACE inhibitor
Blood Pressure	< 160 mm Hg	ACE inhibitor	Amlodipine
IRIS CKD STAGE 2			
Phosphorus	< 4.5 mg/dL	Renal diet	Phosphate binders
UPC	< 0.5	Renal diet	ACE inhibitor
Blood Pressure	< 160 mm Hg	ACE inhibitor	Amlodipine
Bicarbonate	18–24 mmol/L	Correct dehydration	Sodium bicarbonate, potassium citrate
<i>Notes: ARBs may be beneficial</i>			
IRIS CKD STAGE 3			
Phosphorus	< 5 mg/dL	Renal diet	Phosphate binders
UPC	< 0.5	Renal diet	ACE inhibitor
Blood Pressure & Bicarbonate	Same as IRIS Stage 2		
<i>Notes: Use ACE inhibitors and ARBs with caution</i>			
IRIS CKD STAGE 4			
Phosphorus	< 6 mg/dL	Renal diet	Phosphate binders
UPC, Blood Pressure, & Bicarbonate	Same as IRIS Stage 2		

ACE = angiotensin-converting enzyme; ARB = angiotensin receptor blocker; UPC = urine protein:creatinine

- **H2-receptor antagonists:** Require dose adjustment with renal impairment; are less effective in neutralizing gastric pH.
- **Sucralfate:** Helps facilitate GI ulceration healing; may impair absorption of numerous drugs and should be administered alone and without food.
- **Antiemetics:** May be given as needed or as daily therapy.

Hyperphosphatemia

Plasma phosphorus concentrations are inversely proportional to GFR; therefore, as renal function declines, phosphate retention occurs. Hyperphosphatemia increases the production of parathyroid hormone (PTH) by the parathyroid glands, one of the key steps in development of renal secondary hyperparathyroidism.

→THERAPEUTIC GOAL: Treat Hyperphosphatemia

Phosphate binders are used in combination with a prescription renal diet when diet alone is insufficient to control hyperphosphatemia, and form nonabsorbable complexes with dietary phosphate within the GI tract.

- **Aluminum hydroxide:** Often used as first-line drug; however, toxicity has been reported in dogs when administered above recommended doses.
- **Calcium salts:** Must be avoided in patients with hypercalcemia and used cautiously in those with calcium-phosphorus products significantly exceeding 70.

NUTRITIONAL THERAPY is a cornerstone of CKD management.

Prescription renal diets typically have:

- Reduced protein, phosphorus, and sodium concentrations
- Increased B-vitamins, fiber, and omega-3 fatty acids.

Prescription renal diet reduced the risk of uremic crisis by 72% in study dogs when compared to those fed a maintenance diet.¹ Other benefits demonstrated by this study included prolonged median survival time, slower progression of CKD, and improved quality of life.

Lower protein diets, such as senior diets, often do not have the appropriate alterations in phosphorus and electrolyte concentrations recommended for management of CKD; therefore, these diets should NOT be considered acceptable alternatives.

A future article in the Nutrition Notes column (see page 60) will address dietary therapy for renal disease in dogs and cats.

- **Lanthanum carbonate:** Compounding may be required to obtain appropriately sized capsules; can be used in combination with aluminum hydroxide (dose of latter may need to be decreased due to synergistic effects).
- **Sevelamer hydrochloride:** Expands when it contacts water; tablets or capsules should be administered intact. Dose of phosphate binders can be titrated up to produce more pronounced effects. Generally, the more severe the hyperphosphatemia, the higher the dose (kept within the recommended dosage range) of phosphate binder required for successful correction. Treatment should be targeted to achieve recommendations according to IRIS CKD stage. These drugs must be administered with food; feeding meals or phosphate-rich treats without using a phosphate binder lessens their efficacy.

Acidemia

Patients with CKD have metabolic acidosis due to accumulation of acidic uremic toxins; patients with hypoperfusion may additionally have lactic acidosis. If venous blood gas assessment is unavailable to evaluate patient acid-base status, a serum total carbon dioxide level (TCO₂) can be used as an estimate of serum bicarbonate concentration. Falsely decreased TCO₂ levels occur when blood collection tubes are exposed to air or are not fully filled.

→ THERAPEUTIC GOAL: Treat Acidemia

Feed a diet that produces a neutral pH, which prescription renal diets are designed to achieve (but not a feature of some urolithiasis diets). Use alkali therapy (Table 5) for patients with persistent acidemia despite appropriate diet. The goal is to maintain a bicarbonate (TCO₂) level between 18 and 25 mmol/L.

- **Sodium bicarbonate:** Administer as a whole tablet as some dogs find it unpalatable when mixed with food.

TABLE 5. Treatment Goals & Medications for Canine CKD

→ Manage GI Complications of Uremia	
Famotidine	0.25–0.5 mg/kg PO Q 12 H
Maropitant	2 mg/kg PO Q 24 H
Metoclopramide	0.2–0.4 mg/kg PO Q 8 H
Omeprazole	0.1–0.2 mg/kg PO Q 24 H
Ondansetron	0.2–1 mg/kg PO Q 8–12 H
Sucralfate	250–1000 mg/dog PO Q 8 H
→ Treat Hyperphosphatemia	
Aluminum hydroxide	60–90 mg/kg/day PO divided
Calcium acetate	60–90 mg/kg/day PO divided
Calcium carbonate	90–150 mg/kg/day PO divided
Lanthanum carbonate	60–90 mg/kg/day PO divided
Sevelamer hydrochloride	30–135 mg/kg/day PO divided
→ Treat Acidemia	
Potassium citrate	30–60 mg/kg/day PO divided
Sodium bicarbonate	40–60 mg/kg PO Q 8–12 H
→ Treat Anemia	
Darbepoietin	0.45 mcg/kg SC Q week until low normal PCV; then taper dose
Iron dextran	10–20 mg/kg IM, supplemented monthly
→ Treat Hypertension	
Enalapril or benazepril	0.25–1 mg/kg PO Q 12 H; use higher dosages for proteinuria
Amlodipine	0.2–0.5 mg/kg PO Q 24 H
→ Treat Proteinuria	
Enalapril or benazepril	0.25–1 mg/kg PO Q 12 H; use higher dosages for proteinuria
EPA + DHA	40 mg/kg/day divided 25 mg/kg/day divided
Losartan	0.125–0.25 mg/kg PO Q 24 H
→ Treat Secondary Renal Hyperparathyroidism	
Calcitriol	2–3 ng/kg PO Q 24 H

- **Potassium citrate:** Each 540-mg tablet yields 5 mEq of potassium and 1.7 mEq of citrate, which is metabolized to 420 mg of bicarbonate.

While potassium citrate provides some potassium supplementation, which is beneficial to hypokalemic patients, it may exacerbate hyperkalemia in patients with normal or mildly increased serum potassium concentrations. In addition, angiotensin-converting enzyme inhibitor (ACE) inhibitor therapy may also result in mild to moderate hyperkalemia. *Use potassium supplementation cautiously in patients receiving such medications, and avoid use in hyperkalemic patients.*

Hypokalemia & Hyperkalemia

Hypokalemia is more commonly seen in cats than in dogs. Severe hyperkalemia may be life threatening, and is

more often associated with oliguric or anuric acute kidney injury, rather than CKD.

Patients with end-stage CKD and marked reduction in GFR may also demonstrate hyperkalemia, regardless of degree of urine output. By inhibiting the production of angiotensin II, which causes urinary potassium excretion, ACE inhibitor drugs may also produce mild to moderate hyperkalemia as a side effect.

→THERAPEUTIC GOAL: Treat Hypokalemia or Hyperkalemia

- For **hypokalemia**, oral supplementation is the preferred treatment.
- For mild **hyperkalemia**, a prescription renal diet with the lowest potassium content can be useful. Oral potassium binders (sodium polystyrene) can prevent absorption of dietary potassium. Rare GI adverse effects are reported in humans and are possible in dogs.
- *Monitor hyperkalemic patients receiving ACE inhibitors; reduce dose if ACE inhibitors produce significant hyperkalemia.*
- *Discontinue potassium supplementation in all hyperkalemic patients.*

Anemia

Lack of erythropoietin is the driving force behind the chronic, progressive, nonregenerative anemia of CKD. Always consider GI ulceration resulting in blood loss if CKD patients have new or worsened anemia.

→THERAPEUTIC GOAL: Treat Anemia

- For **moderate to advanced anemia** (packed cell volume [PCV] \leq 20%):
 - » Consider hormone supplementation with darbepoietin.
 - » Monitor PCV weekly until target PCV is obtained; then taper frequency of administration.
 - » *Monitor blood pressure as some patients may develop hypertension after initiation of darbepoietin therapy.*
- For **severe anemia**, proceed with a blood transfusion.

Darbepoietin, a synthetic form of erythropoietin, is thought to be less antigenic than human erythropoietin, which can cause development of anti-erythropoietin antibodies that crossreact and potentially destroy the patient's endogenous erythropoietin, leaving the patient dependent on transfusions. Failure to respond to darbepoietin may indicate formation of antidarbepoietin/anti-erythropoietin antibodies; however, concurrent inflammatory disease can also result in diminished response to darbepoietin.

Hypertension

Blood pressure is routinely evaluated throughout treatment of CKD. Normotensive patients may develop hypertension as renal disease progresses. Ideally, assess blood pressure early in the visit before additional stress accrues, leading to nonpathologic increases in blood pressure ("white coat hypertension"). Perform fundic examination to investigate for retinal damage.

→THERAPEUTIC GOAL: Treat Hypertension

- **ACE inhibitors** are first line therapy for hypertension, and crucial to blunting the renin–angiotensin–aldosterone system (RAAS); however, they are weak antihypertensives, only reducing blood pressure approximately 10 mm Hg.
- The **calcium channel blocker** amlodipine is more effective, but should be used with an ACE inhibitor.

Following initiation or increase of ACE inhibitor dosage, mild increases in BUN and creatinine may be noted. *Monitor mild increases that do not cause uremia; however, reduce or discontinue the dosage if azotemia, accompanied by uremia, significantly increases, which suggests the ACE inhibitor has caused a significant decrease in GFR.*

Proteinuria

Renal protein loss may be due to glomerular or tubular lesions, but glomerular lesions more likely result in greater magnitude of proteinuria and hypoalbuminemia. Proteinuria is a risk factor for progression of CKD; however, only weak evidence suggests that reducing proteinuria slows progression of canine CKD.

→THERAPEUTIC GOAL: Treat Proteinuria

The first step in therapy is a protein-restricted renal diet. In addition, managing hypertension also helps minimize proteinuria. ACE inhibitors may cause hyperkalemia due to RAAS blockade, reduce GFR, and increase azotemia; therefore, use these drugs cautiously in IRIS CKD stage 3 and 4 patients.

For persistent proteinuria, therapeutic intervention is recommended:

- **ACE inhibitor:** Increase hypertension dosage to help minimize proteinuria; however, contraindicated in hypotensive or dehydrated patients.
- **Omega 3-polyunsaturated fatty acids:** Shown to lessen proteinuria.
- **Losartan:** Consider this angiotensin receptor blocker for proteinuria refractory to ACE inhibitors; veterinary use has been limited, with contradicting opinions regarding efficacy.

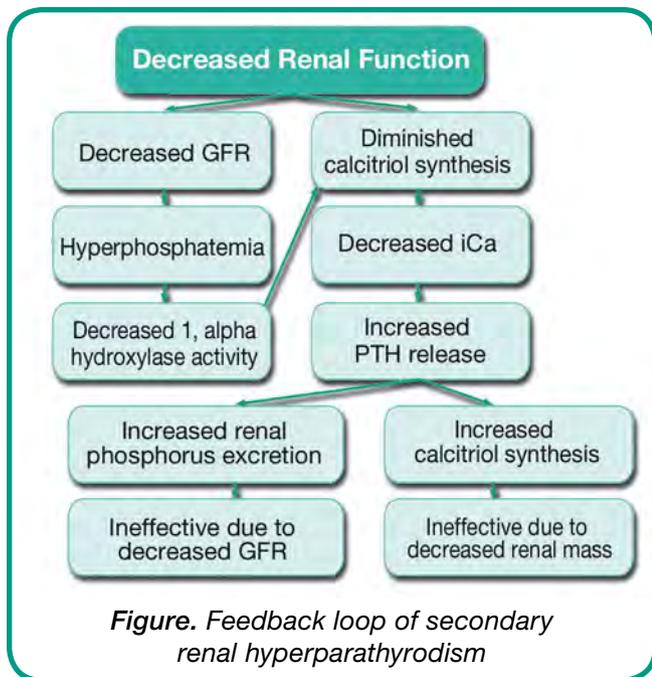
Anticoagulants can be considered when proteinuria is present; however, serum albumin, UPC, or antithrombin levels do not adequately predict hypercoagulability.

Renal Secondary Hyperparathyroidism

Consequences of CKD, including phosphorus retention and decreased synthesis of calcitriol, establish renal secondary hyperparathyroidism.

1. In response to hyperphosphatemia, parathyroid glands increase PTH—a uremic toxin—synthesis
2. Calcitriol inhibits PTH release, but hyperphosphatemia inhibits synthesis of calcitriol, creating a feedback loop that results in elevated phosphorus and PTH levels (**Figure**).
3. Due to decreased renal function, PTH activity is diminished, resulting in inadequate excretion of phosphorus and suboptimal production of calcitriol.

Serum calcium is regulated by PTH; however, normal calcium handling does not occur in renal secondary hyper-



parathyroidism due to altered renal handling of calcium, deficiency in calcitriol production, and skeletal resistance to the effects of PTH. While most dogs with CKD have normal to decreased ionized calcium concentrations, about 15% have ionized hypercalcemia. *Serum total calcium is often discordant with ionized calcium measurements; therefore, do not use it to predict levels of ionized calcium.*

→THERAPEUTIC GOAL: Treat Renal Secondary Hyperparathyroidism

Because calcitriol increases GI calcium and phosphorus absorption, make sure to achieve tight phosphorus control before initiating calcitriol therapy.

- Control hyperphosphatemia to achieve IRIS CKD stage goal (Table 5).
- Then measure PTH and ionized calcium to document inappropriate PTH levels and low or normal ionized calcium concentration.
- Begin calcitriol therapy; administer on an empty stomach.
- Monitor monthly for hyperphosphatemia, hypercalcemia, and alterations in renal function.

MONITORING

Follow-up care is one of the most important aspects of a successful treatment plan.

- As CKD progresses, determine whether new treatments or dosage adjustments to current medications are needed.
- Evaluate physical condition, blood pressure measurement, urinalysis (with sediment evaluation), renal values with electrolytes, and PCV every 4 months minimum.
- Perform urine culture whenever CKD acutely worsens to investigate for pyelonephritis. Due to obligatory poly-

uria in CKD, patients can have an active infection without lower urinary symptoms.

- Effect of therapeutic interventions should be monitored, and medications adjusted to achieve IRIS CKD stage goal.

PROGNOSIS

Prognosis is associated with severity of disease. Studies have shown shorter median survival times in dogs with higher IRIS stages. Median survival time for IRIS Stage 1 dogs was over 400 days, Stage 2 ranged from 200 to 400 days, Stage 3 ranged from 110 to 200 days, and Stage 4 ranged from 14 to 80 days.^{2,3} Successful treatment of CKD delays disease progression, likely provides greater survival times, and increases patient quality of life. ■

ACE = angiotensin-converting enzyme; BUN = blood urea nitrogen; CKD = chronic kidney disease; GFR = glomerular filtration rate; GI = gastrointestinal; iCa = ionized calcium; IRIS = International Renal Interest Society; PCV = packed cell volume; PTH = parathyroid hormone; RAAS = renin-angiotensin-aldosterone system; TCO₂ = total carbon dioxide; UPC = urine protein:creatinine

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Suggested Reading

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Resource

International Renal Interest Society webpage: iris-kidney.com



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