

Chronic Kidney Disease – Canine

David J. Polzin, DVM, PhD, DACVIM

Definition

Kidney disease is the presence of functional or structural abnormalities in one or both kidneys. *Chronic kidney disease* (CKD) is kidney disease that has been present for 3 months or longer. Dogs with CKD and stable serum creatinine values (SC) < 1.4 mg/dL are considered CKD stage 1; SC values between 1.4 and 2.0 mg/dL are CKD stage 2; SC values between 2.0 and 5.0 mg/dL are CKD stage 3; and SC values > 5.0 mg/dL are CKD Stage 4.

Key Diagnostic Tools and Measures

Two to three determinations of serum creatinine, urea nitrogen, phosphorus, bicarbonate, calcium, potassium, and albumin concentrations; urinalysis and urine protein to creatinine ratio (UPC); arterial blood pressure; and assessment of hydration; diet history including food intake; body weight; body condition scoring are used in the diagnosis of CKD.

Pathophysiology

Loss of nephrons leads to waste product retention (especially nitrogenous wastes), impaired body fluid and electrolyte balance, and limited production of renal hormones (e.g., erythropoietin, calcitriol). Markedly impaired kidney function leads to uremia with anorexia, weight loss, and gastrointestinal signs. Excess intakes of protein, phosphorus, sodium, and acidifying nutrients may promote uremic signs and/or progressive kidney damage.

Signalment

Chronic kidney disease typically is found in dogs greater than 7 years of age, although it can occur in all age groups. Breeds predisposed to familial CKD include beagle, English foxhound, bull terrier, Doberman pinscher, Samoyed, Bernese mountain dogs, Brittany spaniel, Rottweiler, Shih Tzu, Lhasa Apso, Keeshond, Norwegian elkhound, cairn terrier, West Highland white terrier, soft-coated wheaten terrier, Basenji, English cocker spaniel, and Shar-Pei.

Key Nutrient Modifications

Limited amounts of high-quality **protein** are indicated in CKD stages 3 and 4. Excess protein intake promotes clinical signs of uremia. Protein intake should also be limited with protein-losing nephropathies. High protein intake does not cause CKD. Limiting dietary **phosphorus** intake slows progression of CKD; this is indicated in stages 2 through 4 CKD to meet serum phosphorus concentration targets (i.e., keep serum phosphorus below 4.5, 5.0, and 6.0 mg/dL in CKD stages 2, 3, and 4, respectively). Phosphorus intake can be limited further with intestinal phosphate binders. Diets enhanced in **omega-3 polyunsaturated fatty acids** (PUFA) slow progression of kidney disease. Excess omega-3 PUFA supplementation may impair coagulation and the immune system and increase the need for antioxidant support. Limit **sodium** intake. Excess salt intake may promote progressive kidney injury and limit hypertension (limited evidence). Excessive salt restriction may promote hypokalemia and dehydration. With regard to **energy**, higher caloric density facilitates ingestion of sufficient food to maintain appropriate body weight.

Recommended Ranges of Key Nutrients

| Nutrient | % DM | g/100 kcal | % DM | g/100 kcal |
|------------|---------------------------|------------|------------------------------|------------|
| | Recommended dietary level | | Minimum dietary requirement* | |
| Phosphorus | 0.2–0.4 | 0.04–0.08 | 0.5 | 0.14 |
| Protein | 14–18 | 3.0–4.5 | 18 | 5.1 |
| Sodium | 0.2–0.3 | 0.04–0.06 | 0.06 | 0.02 |

Modified intake of these nutrients may help address metabolic alterations induced by disease states. The recommended dietary composition is shown as percent of dietary dry matter (DM) and as g or mg per 100 kcal metabolizable energy. All other essential nutrients should meet normal requirements adjusted for life stage, lifestyle, and energy intake.

*Nutrient requirement for adult animals as determined by the Association of American Feed Control Officials

Therapeutic Feeding Principles

The goals of nutritional management of CKD are to 1) ameliorate signs of uremia, 2) maintain electrolyte and acid–base normalcy, 3) optimize nutrition, and 4) slow progressive loss of kidney function. Nutritional therapy using manufactured diets formulated for dogs with CKD has been shown to substantially extend lifespan and limit clinical signs of uremia in randomized controlled clinical trials in dogs with spontaneous CKD stages 3 and 4. Based on results of these clinical trials, nutritional therapy is indicated for dogs with CKD stages 3 and 4. Nutritional therapy may also be indicated for dogs with CKD stage 2 when serum phosphorus remains above 4.5 mg/dL. Because limiting protein intake and providing omega-3 PUFA may limit proteinuria, renal diets are recommended when UPC ratios exceed 2.0 in CKD stage 1 and 0.5 in CKD stage 2. The exact contribution of individual diet components to the beneficial effects of these diets remains to be established. Renal diets should contain ~14% to 17% protein, ≤0.3% phosphorus, and ~0.2% to 0.3% sodium. In addition, manufactured renal diets may also include increased omega-3 PUFA, fiber, vitamin D, and antioxidant contents, increased caloric density, and a neutral effect on systemic pH.

■ **Treats** – Most foods low in protein, phosphorus, and sodium may be used for treats in limited quantities. Kibbles of dry manufactured renal diets are a good choice. Avoid meats and dairy products. In general, treats should constitute no more than 5% of the patient's caloric intake. Commercial dog treats may be used, but only in very limited quantities as they may be high in sodium and phosphorus. Excessive intake of protein-rich treats may induce uremic signs in dogs with stage 4 CKD.

■ **Tips for Increasing Palatability** – Change from the previous diet to the renal diet should be gradual over 7 to 10 days by progressively adding the renal diet to the previous diet. Do not expose patients to long-term diets during periods of hospitalization for uremia. Enhance palatability by mixing small amounts of flavoring (gravy, low-sodium broth) or highly odorous foods into the renal diet. Warming food and stimulating eating by positive reinforcement with petting and stroking may facilitate food acceptance. Avoid associating unpleasant activities (e.g., undesirable medications, fluids) with feeding. When these methods fail to stimulate adequate food intake to maintain body weight, esophagostomy or gastrostomy tubes are indicated. Appetite stimulants rarely produce adequate food intake.

■ **Diet Recommendations** – Manufactured therapeutic diets designed specifically for patients with CKD are recommended. Senior diets are not recommended for these patients because they fail to include all dietary modifications. Dogs should initially be fed 132 x (body weight in kilograms)^{0.75} calories per day. Thereafter, serially monitor body weight and body condition score (BCS) and adjust caloric intake to maintain BCS between 4/9 and 5/9 (see Appendix I).

Client Education Points

- CKD is irreversible and will be present for the remainder of the pet's life.
- Most treatment recommendations for dogs with CKD will need to continue for the remainder of the pet's life.
- Regular follow-up visits are essential to detect changes in treatment needs.
- CKD is usually a progressive disease; however, proper treatment and monitoring can slow progression. Some dogs live many months to years with a good quality of life.
- Free access to fresh water at all times is essential. Never limit water intake.
- Limit excess protein and phosphorus intake (e.g., meats and dairy products).
- Limit intake of salt.
- Progressive weight loss must be addressed to avoid slow starvation.
- Minor gastrointestinal upsets may cause kidney function to abruptly worsen; treatment to prevent dehydration may be needed.
- High amounts of protein in urine and high blood pressure are harmful to the kidneys and require life-long therapy.

Common Comorbidities

Excess sodium intake promotes extracellular fluid volume expansion; excesses in calcium and phosphorus intake may promote vessel mineralization. Both have potential to promote **hypertension**. **Dental and other oral diseases** may be exacerbated by excess protein intake. Inadequate management of dental and oral diseases may impair food intake. In dogs with concurrent **urolithiasis**, renal diets are indicated because of strong evidence supporting renal protective effects while they may also be a reasonable choice for calcium oxalate uroliths. Struvite uroliths are usually of infectious origin rather than dietary. In dogs with concurrent **degenerative joint disease**, weight management and nutraceuticals are preferred over use of nonsteroidal anti-inflammatory drugs due to their potential to harm the kidneys. Finally, the high fat content of renal diets may promote **pancreatitis** in predisposed patients.

Interacting Medical Management Strategies

Intestinal **phosphate binders** (e.g., aluminum, calcium, or lanthanum salts) have an additive effect with dietary phosphorus restriction in reducing phosphorus intake. **Calcitriol** (dihydrocholecalciferol), which is produced by the kidneys and often deficient in patients with CKD, is often used to further suppress renal hyperparathyroidism and slow progression of CKD. Excess dietary vitamin D content may promote hypercalcemia in patients receiving calcitriol. **Angiotensin-converting enzyme (ACE) inhibitors** appear to have a greater salutary effect than protein restriction on mitigating glomerular hyperperfusion, proteinuria and, presumably, progression of CKD. It is unclear whether their effects on renal perfusion and proteinuria are additive. Metabolic acidosis reportedly impairs nutrition in humans receiving protein-restricted diets. **Oral sodium bicarbonate** is used to mitigate metabolic acidosis. As a non-chloride-containing sodium salt, it is not contraindicated with sodium-restricted diets or in hypertensive patients. **Corticosteroids** may impair the nutritional response to protein-restricted diets, enhance proteinuria, and promote gastrointestinal bleeding in dogs with CKD; use sparingly and with great caution. **Erythropoietin** therapy increases patient strength and appetite by increasing hematocrit. Iron supplementation and adequate protein and calorie intake is essential to maximize therapeutic response.

Monitoring

Food intake (food diary), body weight, BCS, serum chemistries (at least serum creatinine, urea nitrogen, phosphorus, calcium, albumin, potassium, and bicarbonate concentrations), hematocrit, and urinalysis (and urine protein:creatinine ratio if proteinuric) should be monitored within the first month after dietary intervention and every 3 to 4 months thereafter. Nutritional status and renal function should remain stable or improve. Ratio of BUN:creatinine should be less than 20; phosphorus should be less than 4.5, 5.0 or 6.0 mg/dL for CKD stages 2, 3 and 4, respectively. Steps should be taken to increase food intake in patients that fail to maintain stable, adequate nutritional status (including use of feeding tubes as indicated).

Algorithm – Nutritional Management of Canine Chronic Kidney Disease

