

Dietary therapy for chronic kidney disease in cats and dogs

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Of all the treatments for chronic kidney disease (CKD), dietary modification has the most positive long-term effect on outcome. With the introduction of the symmetric dimethyl arginine test for CKD, earlier diagnosis and nutritional therapy treatment may improve outcomes even more.

Hydration



Figure 1. Some cats enjoy drinking running water.

Unrestricted access to water is essential in CKD patients to compensate for solute diuresis. Older pets, especially cats, do not sense thirst well and need encouragement to take in enough fluids to prevent dehydration. Provide easy access to water – some cats like running water (**Figure 1**).

Cat water bowls should also be full so the cat can lick from the top without getting its face or whiskers too far into the bowl. Adding water to the food may be beneficial and some pets will eat a soup-like consistency. Some pets benefit from intermittent intravenous or subcutaneous (SQ) crystalloid fluids (Korman and White, 2013) and some owners can learn to provide SQ fluids at home.

Protein and azotaemia

While dietary protein restriction has long been advocated for the management of CKD, the optimal amount of protein for patients with kidney disease is not really known as protein-restricted diets are also usually also phosphorus restricted and the effects of the two parameters are difficult to separate.

Controlled restriction of non-essential protein reduces the accumulation of the nitrogenous waste products, which contribute to the uraemic syndrome and may also decrease acidosis. In one study, cats in International Renal Interest Society (IRIS) stage two or three fed a diet for renal disease with lower protein and phosphorus, showed decreased serum urea nitrogen and increased blood bicarbonate, but there was no difference in serum creatinine, potassium, calcium, parathyroid hormone concentration or urine protein to creatinine (UPC) ratio.

The cats on the diet for renal disease showed fewer uraemic episodes and less renal disease-associated deaths (Ross et al, 2006). Lower protein diets have been thought to slow the progression of renal disease, but this is unproven in dogs and cats.

Proteinuria

Decreased dietary protein is advised for dogs and cats with proteinuria in any IRIS stage of kidney disease as the proteinuria itself is damaging to the kidneys.

Excessive protein in the glomerular filtrate may contribute to additional glomerular and tubulointerstitial lesions and lead to loss of more nephrons. Proteinuric renal disease and systemic hypertension often coexist, and it can be difficult to separate effects of high systemic and intraglomerular pressures and proteinuria.

Persistent proteinuria is at least two increased measurements of increased UPC ratio. The recommendation is to treat persistent renal proteinuria with a renal diet that has reduced dietary protein, phosphorus and sodium, supplemented with omega-3 fatty acids and alkalinising agents, and an angiotensin-converting enzyme inhibitor.

Borderline, and even high, normal levels of proteinuria in cats have been associated with poor outcomes. For example, in cats with naturally occurring CKD, relatively mild proteinuria (UPC ratios of 0.2 to 0.4) increased the risk for death or euthanasia 2.9-fold compared with cats with UPC ratios of less than 0.2 (Syme et al, 2006).

In a study of non-azotaemic cats older than nine years with CKD, proteinuria at presentation (median UPC of 0.19 versus 0.14) was significantly associated with development of azotaemia (Jepson et al, 2009).

As proteinuria is an important risk factor for the development of azotaemia in cats and the progression of CKD in dogs and cats, it should be monitored closely with repeated UPC ratio measurements.

Protein restriction

Higher protein diets do not appear to increase the risk of developing kidney disease. Recommendations for protein restriction in cases with overt signs of CKD are 28% to 35% dry matter basis (DMB) for cats and 14% to 20% DMB for dogs (Forrester et al, 2010).

Renal secondary hyperparathyroidism and phosphorus



Figure 2. Rubber jaw, the decreased mineralisation of bone due to secondary hyperparathyroidism.

Renal hyperparathyroidism (RHPTH), an elevation in parathyroid hormone (PTH), is implicated as a cause of intrinsic progression of CKD, as well as contributing to uraemia. Excess PTH causes demineralisation of bone, leading to the various changes termed renal osteodystrophy (for example, rubber jaw; **Figure 2**).

As the failing kidneys are less able to excrete phosphate, PTH increases in an attempt to increase phosphate excretion. Similar to serum creatinine reference ranges, serum phosphorus reference ranges vary among laboratories.

Cats with stable CKD have lower serum phosphorus concentrations than those cats with progressive CKD. Serum phosphorus concentration is a predictor of CKD progression in cats, with a 41% increase in the risk of progression for every 0.3mmol/l increase in serum phosphorus concentration (Chakrabarti et al, 2012). In 80 client-owned cats with CKD, serum phosphorus concentrations correlated with renal interstitial fibrosis (Chakrabarti et al, 2013).

Initial treatment of RHPTH is dietary phosphate restriction. Recommended phosphorus concentrations in the diet are usually 0.3% to 0.6% DMB for cats and 0.2% to 0.5% DMB for dogs (Forrester et al, 2010). Dietary modification using commercially available renal diets (with phosphate restriction) has been proven to significantly increase survival in CKD (Elliot et al, 2000; Ross et al, 2006).

Many pets in stage two will have plasma phosphate concentrations within the reference range, but will have increased plasma PTH concentration. The elevated PTH initially helps keep the phosphorus within the reference range.

Evidence suggests chronic reduction of phosphate intake to maintain a plasma phosphate concentration below 1.5mmol/l (but not less than 0.9mmol/l) is beneficial to patients with CKD. Some pets, especially cats, with concurrent disease may have low phosphorus and a phosphate restricted renal diet is inappropriate for these pets – that is, assess the pet rather than just reaching for a package with the same name as the diagnosis.

If plasma phosphate concentration remains above 1.5mmol/l (or possibly 1.6mmol/l in stage three patients) after dietary restriction, enteric phosphate binders (for example, aluminium hydroxide, aluminium carbonate, calcium carbonate, calcium acetate or lanthanum carbonate) should be given to effect.

Starting doses are 30mg/kg/day to 60mg/kg/day in divided doses mixed with food at each meal. These medications are of no value unless the pet is eating, as they bind the phosphate in the food.

The dose required will vary according to the amount of phosphate being fed and the stage of kidney disease. Signs of toxicity of phosphate binders limit the upper dose rate. Microcytosis and/or generalised muscle weakness suggests aluminium toxicity if using an aluminium-containing binder. If these occur, switch to another form of phosphate binder.

Serum calcium and phosphate concentrations should be monitored every four to six weeks until stable and then every 12 weeks. Combinations of aluminium and calcium-containing phosphate binders may be necessary in some cases to avoid hypercalcaemia (IRIS, 2013). If vitamin D supplementation is used, the use of calcium-containing phosphate binders should be avoided or used with very careful monitoring.

Treatment with an active form of vitamin D (calcitriol or alphacalcidol) has been recommended to

directly inhibit PTH secretion. This should be used only after the serum phosphorus has been controlled to within the reference range.

This group of drugs has a narrow therapeutic index due to the tendency to cause hypercalcaemia – careful monitoring is needed when they are used (Polzin et al, 2008).

If serum calcium is increased when there is an increased serum phosphorus, there is risk of soft tissue mineralisation, including the soft tissue of the kidneys. Soft tissue mineralisation of the kidney causes irreversible nephron damage and is associated with CKD progression in dogs and cats.

Metabolic complications

Common metabolic derangements associated with CKD include hypokalaemia and metabolic acidosis. Hypokalaemia is more common in cats than dogs, and oral potassium supplementation should be used in any cat with persistent hypokalaemia as this has been associated with muscle weakness (especially ventroflexion of the neck) and morphological renal abnormalities.

Recommended oral potassium doses for supplementation are 3meq/kg to 5meq/kg bodyweight per day of potassium gluconate or potassium citrate. Potassium citrate is also alkalinising.

There should be 0.7% DMB to 1.2% DMB in a feline renal diet and 0.4% DMB to 0.8% DMB in a canine renal diet. Sodium bicarbonate is a good alkalinising agent, but does increase the dietary sodium.

Sodium restriction

Increased dietary sodium has been associated with increased azotaemia in one feline study (Kirk et al, 2006) and is thought to contribute to signs of uraemia in people. This concept is still controversial, and a two-year study in older healthy cats did not find any association between salt consumption and glomerular filtration rate (Reynolds et al, 2013).

Recommended sodium levels in cases of feline CKD are thought to be between 0.2 DMB to 0.35 DMB. There is no known association with sodium and hypertension in cats or dogs.

B vitamins

As the B vitamins are water soluble, increased urine output results in an increased loss of these nutrients.

A diet supplemented with B vitamins is recommended, especially in cats, which have an increased

requirement for B vitamins compared to dogs. Most commercial diets for renal disease have increased amounts of B vitamins added.

Antioxidants and omega-3 fatty acids

Renal oxidant stress may play a role in the progression of CKD. Antioxidant dietary supplementation with added vitamins E and C and beta-carotene has shown reduced DNA damage in cats with renal insufficiency (Plantinga et al, 2005).

Omega-3 fatty acids (such as fish oils) modify the inflammatory response and have been shown to be useful in kidney disease, especially glomerulonephritis (Bauer, 2007).

When a cat or dog won't eat a diet

While cats and dogs on commercial diets for renal disease have significantly better survival than those on maintenance diets, some pets will refuse to eat a renal diet.

In one study, only about half of the cats with CKD were on a veterinary therapeutic diet for renal disease (Markovich et al, 2015). Poor appetite or reduced intake was reported by 43% of the cat owners in this study.



Figure 3. Pets should be transitioned to a renal diet after they are home; this inappetent cat is being tempted with fish during its stay in the clinic.

While recommendations for IRIS stage one dogs and cats is the use of a renal diet only in patients with proteinuria, introducing a diet change early improves diet acceptance and may be indicated.

Early use of the diets and a slow transition will help prevent issues with diet acceptance. In one study, the transition ran for six weeks longer (Ross et al, 2006) and having the cat eat 80% of its intake from the renal diet was considered acceptable for the study.

More than 90% of these cats with CKD accepted renal diets when a very gradual transition was used. The transition to the renal diet should be made at home and not in the hospital as hospitalisation has negative effects that can contribute to food aversion (**Figure 3**).

Adding the water from low-sodium, low-protein flavouring agents such as tuna water or low-salt chicken broth can be used to increase the palatability when the cat's intake is insufficient.

If these methods fail, tube feeding or using a home-made diet formulated for feline kidney disease may be tried. Note, nearly all home-made diets in books and on the internet are not complete and balanced for cats with kidney disease (Larsen et al, 2012).

It should be noted many home-made diets using predominantly meat or poultry are quite high in protein and phosphorus. Diets formulated for senior pets are often lower in phosphorus than diets for maintenance and feeding them is another consideration for pets that won't eat a diet for renal disease, especially early in the disease.

Phosphate binders should be added as needed to control serum phosphate.

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