Chronic kidney disease – dietary management role in dogs

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ABSTRACT

Nutrient manipulation is increasingly an important element of palliative care for many chronic diseases in dogs, and is no more important than in the management of chronic kidney disease. Here, diet plays a key role in slowing disease progression and improving clinical signs. This article aims to explain the key evidence-based nutrient modifications employed in therapeutic renal diets and their specific benefits to these patients.

While clear evidence supports feeding therapeutic renal diets, this can be challenging to achieve in patients where the pathophysiology causes anorexia through multiple mechanisms, with patients often presenting as cachexic. This article will also discuss the longer-term nutritional support that can be provided to these patients.

Keywords: nutrition, chronic kidney disease, renal, prescription diet

Chronic kidney disease (CKD) is the third most common cause of death in canines, affecting 15% of dogs older than 10 years (Royal Canin, 2014).

CKD is progressive, with nephron numbers slowly declining over months or years, prompting a compensatory hypertrophy of the remaining functioning nephrons in an attempt to sustain adequate plasma filtration.

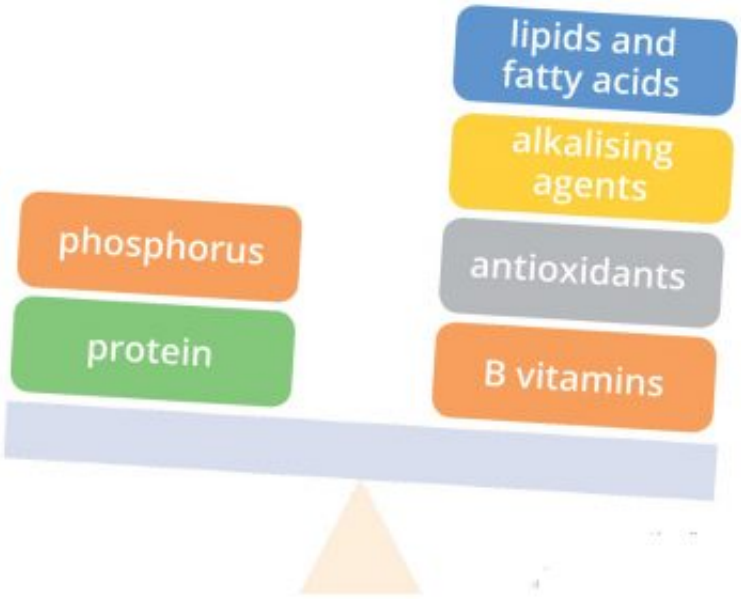

Figure 1. Key dietary modifications for managing canine chronic kidney disease (click to zoom).

As excretory failure worsens, azotaemia, electrolyte imbalances and metabolic acidosis develop – as does, ultimately, uraemia.

Nephron damage in CKD is irreversible and, as such, management of affected dogs is palliative, focusing on easing the distressing physical effects of the disease, as well as slowing its progression by preserving the remaining renal function.

Dietary modification has been recognised as playing a role in managing CKD for 60 years (Roudebush et al, 2010) and is now considered the cornerstone of CKD therapy, achieving the aims of palliation through the modification of key components within the diet (**Figure 1**).

This article will review the main dietary modifications and their specific benefits to dogs with CKD, and look at how to maximise nutritional intake, as well as monitor the adequacy of the diet.

Energy

While not a nutrient, it is vital to consider the energy sources and energy density of any renal diet. Energy is measured in kilocalories (kcal) and is the most important component of any diet.

Energy can be gained from all of the three macronutrients – lipids, carbohydrates and protein – and it is essential a dog with CKD has its energy requirements met.

Failure to do so will result in – to reduce the energy deficit – the catabolism of muscle tissue to extract amino acids.

This breakdown of muscle, in turn, would elevate the levels of urea precursors in the blood, exacerbating azotaemia, as well as compromising muscle function, which would result in increased patient morbidity.

CKD dogs often present cachexic and/or in poor body condition, having already lost bodyweight and muscle mass, and with a low body condition score (BCS; **Figures 2 and 3**).

Prescription renal diets contain higher levels of lipids to increase their energy density and help preserve patients' lean muscle mass.

The energy requirement of a dog with CKD is likely similar to that of a healthy adult dog (Wortinger and Burns, 2015) and this seems a sensible place to start.

Besides, serial nurse clinic visits will be necessary to monitor the nutritional status of the individual, so the kcal requirement can be adjusted based on the results of physical and biochemical assessment of the dog as time goes on (**Table 1**).

Daily kcal requirement for a dog with CKD is often worked out as 132 × bodyweight(kg)0.75 (Aguirre and Darling, 2012).

Low body condition is a common result of canine CKD. IMAGE: Fotolia/Juhku.

Figure 2. This patient with chronic kidney disease presented with a body condition score of two.

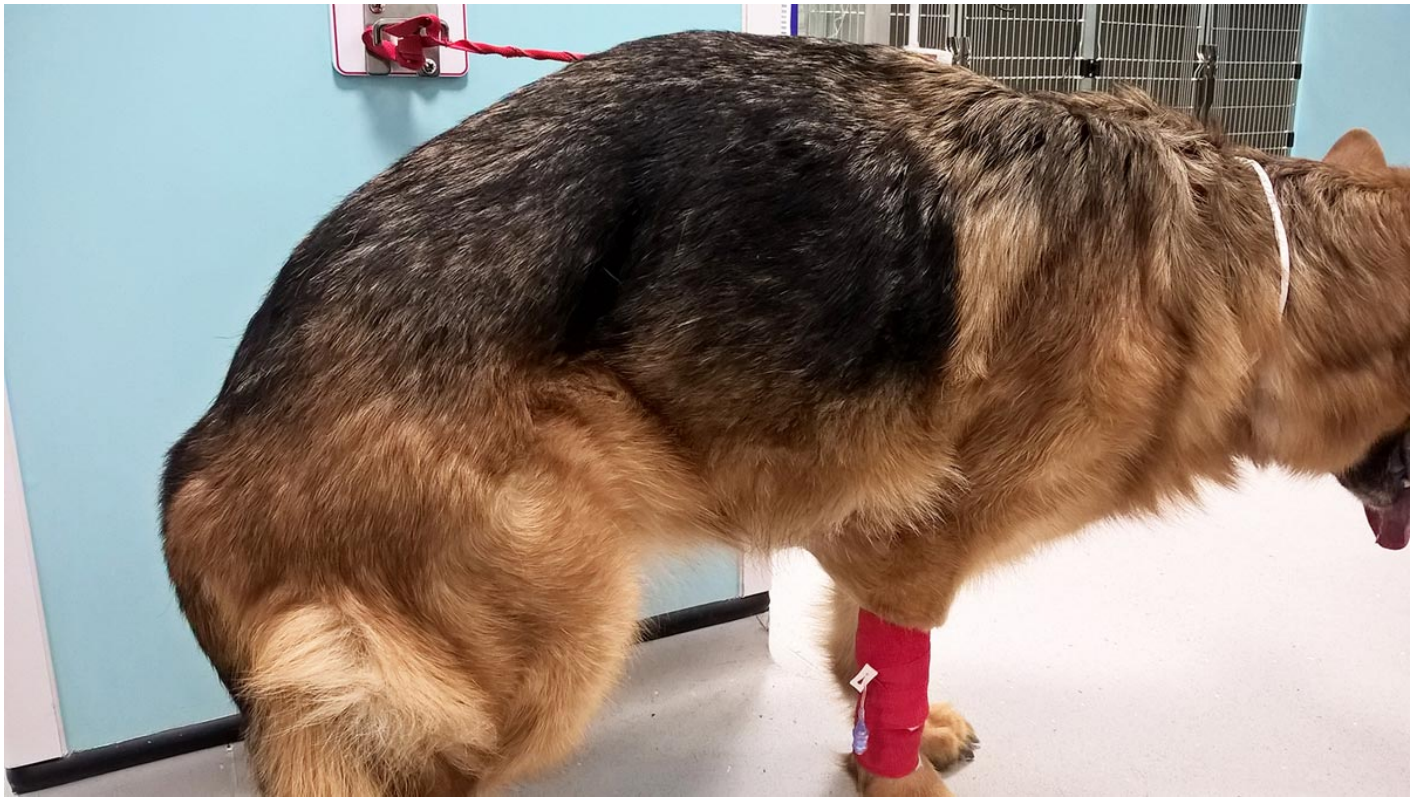

Figure 3. Marked loss of lean muscle mass was apparent on the limbs, lumbar and temporal regions, with an exaggerated abdominal tuck/waist and prominent pelvic bones, dorsal spinal processes and ribs.

Protein

The negative role of excessive protein in CKD has long been recognised as the kidneys are responsible for the filtration and excretion of nitrogenous waste products produced from the deamination of dietary and endogenous amino acids.

Any excretory failure results in azotaemia and later uraemia; biochemical abnormalities responsible for many of the quality-of-life reducing symptoms experienced by CKD dogs, such as nausea, vomiting and anorexia.

Protein has many diverse and vital functions within the body. It is a structural component of all tissue and is used to manufacture immunoglobulins and plasma proteins, such as albumin and haemoglobin.

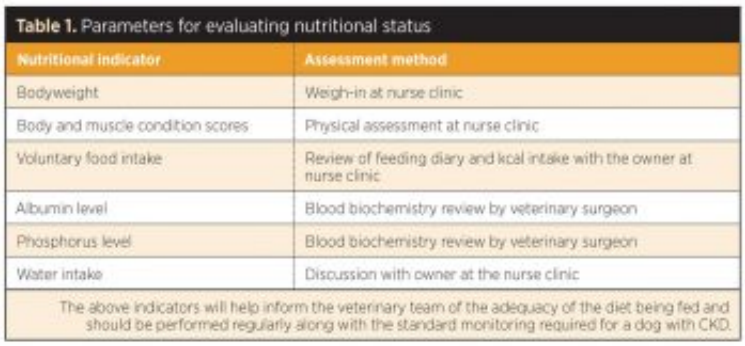

Table 1. Parameters for evaluating nutritional status

Nutritional indicator	Assessment method
Bodyweight	Weigh-in at nurse clinic
Body and muscle condition scores	Physical assessment at nurse clinic
Voluntary food intake	Review of feeding diary and kcal intake with the owner at nurse clinic
Albumin level	Blood biochemistry review by veterinary surgeon
Phosphorus level	Blood biochemistry review by veterinary surgeon
Water intake	Discussion with owner at the nurse clinic

The above indicators will help inform the veterinary team of the adequacy of the diet being fed and should be performed regularly along with the standard monitoring required for a dog with CKD.

Table 1. Parameters for evaluating nutritional status (click to zoom).

The digestion and assimilation of nutrients, meanwhile, requires protein-based enzymes, while the nitrogen element of protein is essential to synthesise many compounds.

The limitation of dietary renal protein can contribute to the reduction of the clinical signs of uraemia in CKD and is usually indicated in stages three and four of the disease. Otherwise, the ideal quantity of protein to be fed to dogs with CKD is unclear (Bartges, 2010).

Clearly, some reduction of dietary protein is required, but protein malnutrition will also impact negatively on the morbidity and mortality of dogs with CKD, and must be avoided.

Prescription renal diets contain high biological value protein in quantities to meet minimum protein requirements when fed in sufficient volumes.

This level of protein quality and reduction, while not definitively proven in dogs to slow disease progression, should ensure all internal protein-dependent processes can continue and is proven to produce clinical benefits to the dog by reducing the magnitude of the azotaemia and its physical manifestation.

In addition, by reducing the amount of solute delivered to the nephrons, the severity of the dog’s polyuria and polydipsia is also decreased (Elliott and Lefebvre, 2010).

In recognition of CKD’s progressive nature, as well as the differing levels of protein tolerance across the spectrum of renal function loss, prescription renal diets are now available that target different stages of CKD.

Phosphorus

Regulation of phosphorus within the body is coordinated by the intestines and kidneys, which are working to control the intestinal absorption and renal excretion of dietary phosphorus. In CKD, the reduction in glomerular filtration rate results in reduced phosphorus clearance and

hyperphosphataemia.

Consequently, levated phosphorus causes renal secondary hyperparathyroidism, reduced levels of calcitriol and renal mineralisation, all of which can contribute to progression of CKD (Bartges, 2010; **Panel 1**).

Diets that restrict phosphate have been shown to slow the decline – and enhance survival – of dogs with CKD, and recommendations for dogs in stages two, three and four include phosphate restriction (Bartges, 2010).

Renal diets provide as low as 0.13% phosphate – much lower than the 1 to 2% in standard diets (Bartges, 2010).

Panel 1: Effects of hyperphosphataemia

1. Secretion of parathyroid (PTH) and development of renal secondary hyperparathyroidism resulting in hypocalcaemia, renal osteodystrophy and further release of PTH
2. Deposition of the phosphate-calcium product results in renal mineralisation and ongoing renal inflammation and fibrosis

Prescription renal diets alone are not always successful in achieving normal phosphate levels in severe cases of CKD, so intestinal phosphate binders, such as aluminium, calcium, lanthanum or sevelamer-based products, should be used for such challenges. These compounds bind with dietary phosphate and digestive secretions, thereby preventing the intestinal absorption of phosphate.

However, their addition to every meal can impact the dog's acceptance of food, reducing overall caloric intake, while their benefit, well-established in improving survival in azotaemic cats (Geddes et al, 2016), has not yet been proven in controlled trials of dogs with naturally occurring CKD.

Antioxidants

Oxygen free radicals are highly reactive molecules missing an electron and their production is elevated in many chronic disease states, of which CKD is no exception.

Their unstable molecular structure results in an interaction with many compounds in the body in an attempt to gain an electron – a process that results in oxidative damage.

Humans with CKD have been shown to have oxidative stress, with the damage caused by oxygen free radicals believed to have a significant role in the progression of the disease (Royal Canin, 2014).

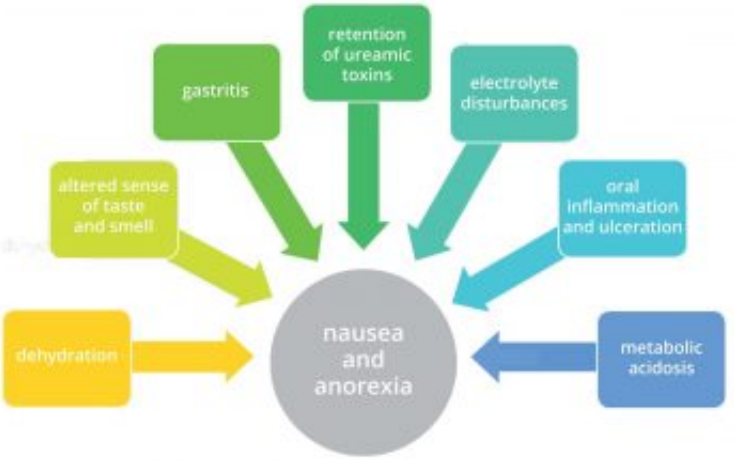

Figure 4. Factors that contribute to anorexia in dogs with chronic kidney disease (click to zoom).

The body has inherent antioxidant systems to scavenge and neutralise free radicals produced under normal circumstances, but it is likely these become depleted in CKD.

The addition of exogenous antioxidants, such as vitamin E, vitamin C and flavanols, to therapeutic diets can help restore the balance and minimise ongoing oxidative damage.

In addition, dogs whose diets are supplemented with omega-3 polyunsaturated fatty acids (PUFA) have lower mortality, better renal function, fewer renal lesions, less proteinuria and a lower cholesterol level when compared to dogs eating a diet high in saturated fats or omega-6 PUFA (Bartges, 2010).

Appetite

Dogs with CKD are twice as likely to suffer from anorexia and inappetence (Royal Canin, 2014) for a variety of reasons (**Figure 4**). To maximise the acceptance of a prescription diet, aversive conditions – such as nausea, dehydration and oral pain – should be controlled first, with the dog in its home environment.

The transition from the dog's usual diet to a diet modified for CKD may be challenging, as taste receptors will be accustomed to certain nutrient levels and will need to adapt to the new ones. This will take time and patience, and success requires owner compliance; unless fully prepared in advance, they can become disheartened, so be wary of this.

A less significant dietary change would be to put the dog on to a senior diet before moving on to a

renal therapeutic diet.

Voluntary intake of food can be encouraged by warming food, hand feeding and feeding small, regular meals. With the increasing variety of renal therapeutic diets now available, owners can experiment with several different food textures and aromas, and should be encouraged to persevere.

Owners may decide to feed a home-prepared renal diet, but one paper (Larsen et al, 2012) evaluating these recipes found them to be nutritionally inadequate with inappropriate and variable levels of nutrients, despite their seemingly valid origins. Such diets should only be employed under the direct supervision of a veterinary nutritionist. Ideally, a moist diet is preferred to help maintain hydration status.

This food is also softer and easier to manipulate, considering the oral pain common in CKD. Owners should be asked – where possible – to avoid adding medication to meals as this too can result in reduced acceptance.

Figure 5. An oesophagostomy tube placed for providing mid-term support to a patient with CKD – control of azotaemia could be achieved and appetite improved.

Extremely challenging anorexics may benefit from appetite stimulants or placement of a feeding tube, with either an oesophagostomy or gastrostomy tube offering longer-term nutritional support (**Figure 5**). Liquid feeds are delivered easily down even narrow bore tubes, and ones modified for CKD exist.

Conclusion

The clinical benefits of a therapeutic renal diet for dogs with CKD have been well established.

These patients can experience reduced symptoms through nutrient modification, but, paradoxically, adequate food intake can be challenging to achieve and even the best formulated diet in the world will have no effect if it is not eaten.

As nurses, our role is to:

- understand the nutrient modifications found in therapeutic renal diets
- understand the benefits they offer to patients
- convey information clearly to owners
- offer long-term support to owners
- provide regular, documented nutritional evaluations of the dog using standardised and appropriate tools

This article was reviewed by Kerry Doolin BSc, BVSc, MVetMed, MRCVS.

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