Effect of amino acid and peptide complex AB070597 on renal function in dogs with chronic kidney disease

The management of chronic kidney disease (CKD) in dogs remains a challenge to veterinary professionals and exerts financial burdens on their owners. Expanded knowledge and improvement in treatment would benefit all. The objective of the present study was to examine whether AB070597, a dietary-supplement compound of six amino acids and one peptide, can slow, halt, or reverse the decline of renal function in dogs with CKD. Five dogs with CKD were enrolled in a longitudinal study over 29 weeks. Animals received bi-daily oral doses of AB070597 from the point of enrolment to the end of the study, and blood samples were taken approximately every four weeks (for a total of 10 time intervals) to measure CKD-relevant biochemical parameters.

Results
The typical decline of renal function reported in dogs with CKD receiving standard palliative care, as measured by significant increases in blood-serum creatinine concentration (SCr), blood urea nitrogen (BUN), blood-serum phosphate concentration (PHOS), and lowered urine specific gravity (USG), was not observed. Median SCr, BUN, PHOS, and USG were stabilised and there was no significant change during any time interval.

Conclusions
These findings suggest that AB070597 may be a useful tool in stabilising or preventing the progression of CKD in some canines. The formulation warrants further study.

Keywords: Chronic kidney disease, renal insufficiency, AB070597, dogs, dietary

Introduction

CKD is the most common kidney disease in dogs[65]. Prevalence, however, varies widely from 0.05[66], 0.9, 0.5-1.5 to 3.74%[68], depending on the source population and inclusion criteria.

CKD is less prevalent in dogs than in cats and the age at onset varies due to a number of breed- related diseases which affect the canine kidney. The mean age of dogs diagnosed with CKD is seven years[69]. Irrespective of disease cause, it is widely accepted that most dogs diagnosed with CKD will progress inevitably to end stage disease and that BUN, SCr, PHOS will increase and USG will decrease significantly[3,4,5].

Many pathways lead to renal disease in humans, dogs, and other mammals. Mammalian renal architectures are similar, and even though there are some metabolic differences, it is possible to draw from the body of scientific information concerning mammalian renal disease, and with appropriate caution, apply those findings in an attempt to improve available treatment options for dogs with renal dysfunction. The most interesting findings in this context are related to disease progression monitoring via SCr, and data that suggests the role of reactive oxygen species (ROS) in renal cell damage.

Certain lipoproteins induce the formation of reactive oxygen species (ROS) in glomeruli and in arteries. Antioxidants may prevent the damaging effects of these lipoproteins[24, 32, 34, 36, 39, 40-43, 47, 55]. Progressive injury results directly and indirectly from angiotensin II receptors via mediators of angiotensin-II-induced renal injury through transforming growth factor-beta, fibroblast growth factor-beta, tumor necrosis factor-alpha and platelet-derived growth factor[5, 6, 7, 11, 25, 31, 54]. In addition, angiotensin increases oxidative stress, which causes a vasoconstrictor effect by increasing the catabolism of nitric oxide (NO)[20, 26, 35, 36]. Aldosterone is also a major contributor to the progression of CKD[26]. All of these compounds promote the progression of CKD by enhancing cell growth, fibrosis and inflammation, which destroy tubulointerstitial tissues and glomeruli[4].

Multiple approaches have been used to prevent the progression of renal disease, such as protein-restricted diets, the control of hypertension with angiotensin converting enzyme inhibitors, diet substitution of saturated fats with polyunsaturated fats, immunosuppressants such as mycophenolate mofetil, corticosteroids such as prednisone[1, 36, 52, 57, 58] and morphogenic agents such a bone morphogenic protein-7 (BMP-7)[64]. None of these treatments hold promise for halting or reversing disease progression, with the exception of BMP-7. Previous studies have shown that BMP-7 can reverse epithelial to mesenchymal transition in murine models of acute renal failure and can promote renal tissue repair[64]. AB070597 is a cytoprotective agent that reduces damage to renal tubules and increases the glomerular filtration rate (GFR), stimulates gluconeogenesis and suppresses proteolysis in skeletal muscle, has strong anti-inflammatory properties, is a precursor for NO production and increases BMP-7 (Archer J, unpublished observations).

Methods and Subjects

An open-enrolment, open-ended study format was selected so that dogs with confirmed CKD could be added periodically. Dogs were confirmed with CKD if 1) their SCr >/= 2.3 mg/dL, 2) their USG was less than 1.050, 3) their BUN was elevated near or above the high end of the normal range, 4) their PHOS was elevated near or above the high end of the normal range and 5) their clinical history included signs attributable to CKD (i.e. persistent azotemia, chronic polyuria and polydipsia, or small kidneys on abdominal palpitation). If one or more of these signs were present with increased SCr, dogs were considered for inclusion in the study. Dogs with suspected or verified conditions, such as pyelonephritis, uncontrolled hypothyroidism, acute renal failure, cancer, or other CKD-unrelated diseases were excluded. The criteria used to establish the cessation of progressive renal injury were 1) a halt in the rise of SCr, BUN and PHOS and a halt to the decline of USG, all for an extended period of time. No statistically significant deterioration in these values would signify no statistically significant disease progression. Dog owners were given informed consent forms for review and acceptance. The amino acids and peptide in AB070597 were purchased from Spectrum Chemical Company (Gardena, California).

Over a 29-week period, five subjects ranging in weight from 3.2 to 18.5 Kg (mean = 7.8, median = 4.7, range = 3.2-18.5 Kg), all on non-protein-restricted commercial diets, received two 1000 mg / 4.5 Kg body weight oral daily doses of AB070597 as a dietary supplement. Doses were mixed with approximately 3

millilitres of water and administered directly into each subject's mouth, or the dose was sprinkled directly on a small amount of food and fed to the subject. AB070597 was readily accepted without rejection. SCr, BUN, PHOS and USG measurements were made for each subject at four-week intervals during the 29-week study (mean = 22, median = 16, range = 16-38 weeks).

Statistical Analysis

Statistical comparisons were made by comparing measured parameters upon entering the study with measured parameters at the end of the study. Values were calculated with the Wilcoxon signed-rank test (VassarStats: Website for Statistical Computation, www.vasarstats.net). Differences were considered statistically significant at $P \leq 0.05$ (two tailed).

Results

Each patient was monitored over the course of the study. Assessments were made for general body condition, weight change and ease of administration of AB070597. There were no owner complaints or concerns regarding administration of the supplement. General body condition, including coat, appearance and grooming habits, improved in each patient, and 60% gained weight, while body weight of the other 40% remained stable without further loss. There were no reports of gastrointestinal upset or diarrhoea. The medians of SCr, BUN, PHOS and urine USG showed no statistically significant change from start to finish Table 1.

Discussion

Measured parameter	Start median	Start IQR	End median	End IQR	P
SCr (mg/dl)	2.0	1.4	1.6	1.9	ns
BUN (mg/dl)	48	54	51	44	ns
PHOS (mg/dl)	3.4	1.6	1.5	2.3	ns
USG	1.015	0.004	1.022	0.006	ns

IQR = Interquartile range

Table 1. Changes in the measured parameters of the sample population over the study duration.

CKD is a progressive disease that results in significantly increased SCr, BUN, PHOS and lowered USG over time. Oral administration of AB070597 attenuated progression, such that there were no significant (ns) changes in those values. A possible hypothesis is that these results are due to biochemical effects of the individual components of AB070597.

L-arginine protects renal tissue from the negative effects of renal ischemia [12, 50, 53, 59] and facilitates the disposal of protein and metabolic waste, and acts globally on muscle metabolism, vascular tone regulation and immune system function, and promotes the release of numerous hormones (glucocorticoids, growth hormone, prolactin, insulin, somatostatins, glucagons, catecholamines) through various pathways, whose disturbance can cause detrimental effects on renal function[2, 3, 4, 18]. L-arginine admini-stration also relieves a variety of pathological states, including kidney hypertrophy and glomerular thrombosis[2, 10, 50, 53].

Glycine is produced in the mammalian body in amounts up to fifty times greater than those taken in daily by diet. Any decrease in its natural production is therefore of concern. The body uses this amino acid to form RNA, DNA porphyrin, bone collagen, glutathione, heme, bile, and salts and for the detoxification and conjugation of toxic products, both exogenous and endogenous. It exerts a cytoprotective effect against anoxia, ischemia, heat, antibiotics, metals, and indomethacin-induced kidney damage. It also increases the GFR and thereby improves kidney function[16, 17, 48].

L-glutamine is the precursor of nucleotides and proteins and is the substrate for and stimulates gluconeogenesis in all organs and tissues. This amino acid regulates carbohydrate metabolism and suppresses proteolysis in skeletal muscles and stimulates protein synthesis, thereby counteracting the muscle-wasting effects caused by CKD[8,21,23,37,38,46,56,61,62]. L- glutamine is also an efficient ROS scavenger[63].

L-histidine concentrates in the brain at a level five times higher than in blood serum. It is readily available from food, but food intake is reduced in animals with CKD, thereby reducing the normal total cerebral amount. It also has anti-inflammatory properties and counteracts the damaging effects of ROS formed by those processes [15, 44].

L-aspartic acid and L-glutamic acid, as sodium salts, are present in all tissues. The highest concentrations are located in the central nervous system. L-aspartic acid sodium is distributed throughout the central nervous system and spinal cord[22]. L-glutamic acid sodium is distributed in the caudate nucleus and cerebral cortex[30]. They act as efficient ROS scavengers and thereby protect the kidneys from ongoing damage caused by these ROS radicals[63].

L-carnosine increases the production of BMP-7[28]. BMP-7 induces mesenchymal-to-epithelial transition in renal fibroblasts and facilitates regeneration of injured kidney[64].

Conclusion

Dogs with CKD treated with AB070597, as a dietary supplement, did not experience increased SCr, BUN, PHOS, or a decrease in USG typically seen in dogs with CKD receiving standard palliative care. Treated animals had stable or reduced values for these parameters for up to 29 weeks. Results suggest that the oral supplement AB070597 may promote the maintenance of renal function and potentially attenuate or prevent progression CKD in dogs. Additional studies with more subjects and longer follow-ups are warranted, and future studies of AB070597 and its effect on renal function in dogs employing a much larger population are planned.

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