International Congress of the Italian Association of Companion Animal Veterinarians

29 - 31 May, 2009
Rimini, Italy

Next Congress:

65th SCIVAC International Congress
May 28-30, 2010 - Rimini, Italy

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Chronic renal failure

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Chronic renal failure represents the irreversible loss of functional capacities of the kidneys as a result of a reduction in the number of functioning nephrons. In the first stage, the remaining nephrons increase their size and their work load to compensate for nephron loss. Nevertheless this chronic effect, with nephron destruction initiates further compensation, promotes a cycle of adaptation, who progress into renal failure, uremic syndrome and death. The end-stage of renal disease is defined as the almost complete failure of renal function or irreversible destruction and is characterized by extensive glomerular sclerosis, tubular atrophy, interstitial inflammation, and fibrosis. Renal fibrosis is a common pathway leading to kidney failure. Infiltrating immunocytes in the end-stage kidney and several related factors are involved in renal fibrogenesis.

CRF is a common clinical problem occurring in 2-5% of dogs over 6.5 years. It is the third leading cause of death in dogs. In cats, the prevalence is 2-10%, up to 53%, over 7 years old. The onset of renal failure tends to be insidious as renal function generally declines over a period of months to years.

Renal function loss has effects over different levels: endocrine system, electrolyte acid-base balance, calcium metabolism and the synthesis of erythrocytes. On the other hand, we have the consequences of high urea concentrations in blood and tissues.

Medical treatment has supported to reduce the work of the functional kidneys, to reduce uremic status, to compensate metabolic disorders, and ultimately slow down the disease. The nutritional therapy is the mainstay of control this disease. The objectives are determined by anorexia, weight loss, proper glomerular filtration, uremic toxins and skeletal abnormalities. Nutrition plays a key role in improving quality of life and life expectancy of these patients retarding progression of the disease. Typical nutritional interventions include modifying the protein, phosphorus, and lipid concentrations. Protein levels are very important for control of azotemia and uremia. The premature control of phosphorus alterations can increase the survival of the patient. This mineral has correlated with disease progression. Some conducted trials have done with different levels of phosphorus and protein, and as conclusion suggests that patients benefit from low levels of these nutrients. Nutritional therapy, however, does not simply mean changing the diet; consideration must also be given to ensuring adequate caloric intake and to the method of feeding. Monitoring the effects of the dietary therapy is also crucial to ensure that the patients are responding appropriately to the selected nutritional modifications. Nutritional management must be coordinated with medical management for long term successful treatment.

Oxidative stress may contribute to the progression of chronic renal failure. Moreover, the incorporation of some specific nutrients such as antioxidants, i.e. supplements of vitamins E and C and beta-carotene, omega 3 and 6 fatty acids exert protective effects on the animal.

Anyway, as the dynamic and progressive nature of this disease, the treatment is not efficient when we are at stage IV, so that the owners requested euthanasia for humanitarian reasons. Earliest diagnosis and treatment assures an acceptable alternative.

References


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