SUCCESSFUL MANAGEMNET OF CHRONIC KIDNEY DISEASE IN DOGS

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Conservative management of advanced stages of CKD in dogs is consisted of a multifaceted approach towards management of clinical signs and correction of fluid and electrolyte imbalances, acid-base status, anemia, hyperphosphatemia, hypertension and proteinuria. An evidence based approach was used in the treatment of IRIS Stage 3 and 4 CKD in 6 dogs for a period of five weeks, to record changes in hemato-biochemical parameters. Successful management of disease with correction of the several imbalances was achieved when therapy has targeted according to the needs of each individual patient. It was thus concluded that targeted conventional medical therapy, along with treatment of any primary kidney disease was imperative in management and improvement of long-term prognosis of CKD in dogs.
Keywords: CKD, Acid-base status, Anemia, Hyperphosphatemia, Hypertension, Proteinuria.

A high prevalence of chronic kidney disease (CKD) has been recorded in humans and dogs alike. Sosnar *et al*., (2003) recorded a prevalence of 11.9%, with a mortality as high as 76.6% in dogs. Treatment for CKD is prolonged due to the progressive nature of the disease, and lifelong management of the disease is warranted. Conventional therapy for CKD consists of staging of disease, correction of hydration status and electrolyte imbalances, management of anaemia, proteinuria, and systemic hypertension and supportive therapy for other clinical signs. Optimization of treatment protocol according to the individual patient for best long-term outcome is warranted. This study was undertaken with the aim to track progression of CKD in 6 dogs over a period of five weeks that were treated according to their CKD staging and clinical signs.

Materials and Methods

The study was conducted on dogs presented at the Teaching Veterinary Clinical Complex, Nagpur Veterinary College, Nagpur. Based on history and physical examination, kidney function testing was performed. Dogs having a Serum Creatinine of > 2 mg/dL and a BUN of > 25 mg/dL were included in this study. Haemato-biochemical parameters estimated were Haemoglobin, Packed Cell Volume (PCV), Blood Urea Nitrogen (BUN), Serum Creatinine, Serum Sodium, Serum Potassium, Serum Chloride, Serum Phosphorus. Urine was collected for routine urinalysis and Antibiotic Sensitivity Testing (AbST). Abdominal ultrasound examination was carried out for all patients. The cases fulfilling the inclusion criteria were incorporated in this study.

Treatment

Intravenous fluids were administered depending on electrolyte imbalance and percentage of dehydration. Antibiotic Sensitivity Testing was performed and antibiotics were administered according to the same, in case of clinical UTI, broad spectrum antibiotics were initiated until the AbST report came in. For the cases which showed hyperphosphatemia, Tab Sevelamer Hydrochloride 50 mg/kg B.W. PO was prescribed till the serum phosphorus levels returned to normalcy(Polzin, 2013). Inj. Darbepoetin @ 0.45 ug/kg B.W. SQ was given once a week to dogs found to be anaemic (Fiocchi *et al*., 2017). For patients whose systolic blood pressure exceeded 180 mm Hg in a clinical setting, therapeutic intervention with ACE inhibitors (e.g. Enalapril @ 0.25-1.0 mg/kg po q 12-24 hours) and/or calcium channel blockers (e.g. Amlodipine @ 0.25-0.5 mg/kg) was undertaken. Other symptomatic treatment

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such as Ondansetron (0.5 mg/kg. B.W.) and Ranitidine (0.5 mg/kg B.W.) were given parenterally or orally when required. The patients were monitored daily for improvement in clinical condition and the treatment regimen was modified as per the requirement. Dietary changes were initiated with slow transition to a prescription renal diet for all patients.

Results and Discussion

A characteristic anemia for Stage 3 and 4 CKD was noted in all cases. A high percentage of patients were presented to the clinic when their disease had already progressed to an advanced stage, with pallor being a common clinical sign. In the present study, patients were treated with Darbepoetin @ 0.45 ug/kg B.W. SQ once a week and Inj. Iron Dextran @ 10 mg/kg at the start of treatment, followed by oral iron supplementation, recorded average haemoglobin of 9.12 ± 0.64 g/dL at Day 0 which showed a steady decrease till Day 14 followed by an increase till Day 28 at 9.03 ± 0.76 g/dL. These values were consistent as also mentioned by Liu and Su, 2015. A similar trend was noted with PCV, various values observed have been depicited in Table -1.

BUN is a nitrogenous waste substance created from catalysis of protein, which is filtered and somewhat reabsorbed at the glomerulus. It is not the most accurate factor to estimate GFR, but fluctuation in level helps determine kidney function. Mean BUN value at the start of the experiment was noted to be 104.08 ± 21.21 mg/dL, which decreased to 76.12 ± 17.82 mg/dL by the end. The overall reduction in BUN could be attributed to fluid therapy tailored to each patient's requirement. In agreement to our study Grauer (1998) also reported that the primary goal of treatment for renal failure was correction of hydration and electrolyte imbalance in order to achieve diuresis and reduce uraemia.

Serum creatinine is considered a better indicator of GFR than BUN due to the fact that it is filtered by the kidney, without being significantly reabsorbed. IRIS guidelines suggested staging of renal disease to be done according to serum creatinine levels as have been also recorded by Anonymous, 2017. In the present study, the mean serum creatinine had a starting value of 8.13 ± 2.03 mg/dL which at Day 28 was decreased to 5.92 ± 1.65 mg/dL, although a slight increase in the value was recorded from Day 21 to Day 28. Reduction in Serum Creatinine post treatment can be attributed to the diuresis, thus increased perfusion to kidneys caused by appropriate fluid therapy.

Loss of normal renal function results in loss of electrolyte and acid base balance, which if uncorrected, can be correlated with increased mortality. Sodium is the primary cation in extracellular fluid. Any change in the serum sodium concentrations is dictated more by the loss or gain of total body water and both hyponatremia and hypernatremia have been recorded in renal failure as also narated by Rosenberg, 2017. The mean serum sodium recorded on Day 1 was 135.48 ± 3.32 mmol/L and 137.15 mmol/L on Day 28. The marginal increase in sodium levels could be attributed to correctional fluid therapy. Potassium is the major intracellular cation of the body. Hypokalemia is more common in patients with clinical signs of CKD due to increased renal loss, complication of which may present as muscle weakness. The mean value of serum potassium was 4.33 ± 0.34 mmol/L on Day 0 which decreased to 4.16 ± 0.22 mmol/L on Day 28. Serum potassium remained in the normal range throughout the duration of the study. In accordence to ourstudy, Ramesh *et al.,* 2018 also recorded serum potassium values in the normal range in CKD affected dogs. The chloride ion absorption, regulation and excretion from the kidney is associated with sodium and water. Chloride and bicarbonate ions together maintain the renal acid-base regulation. The mean value for Serum Chloride on Day 0 was 96.07 ± 3.58 mEq/L, which increased to 101.73 ± 2.46 mEq/L on Day 28.Loss of chloride ions due to vomiting and dehydration could explain the hypochloraemic state of patients, which was corrected with appropriate fluid therapy. Phosphorus is a major intracellular anion whose regulation in the body in controlled by parathyroid

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hormone and Vitamin D. As also reported by Sumit *et al.*, 2018, an increased serum phosphorus on electrolyte estimation in dogs with renal failure. Consequences of hyperphosphatemia are severe. Hyperphosphatemia leads to secondary hyperparathyroidism, bone disorders and soft tissue calcification, and is associated with a poor long-term prognosis. The mean value of serum phosphorus on Day 0 was 7.52 ± 0.93 mg/dL, which reduced to 5.97 ± 0.35 mg/dL on Day 28. In aaccordanceto our study, Polzin 2013 also recommended use of oral phosphorus binders to reduce availability and absorption of dietary phosphorus.

Table 1 Mean ± SE values of Haemato-biochemical parameters of patients in the study, recorded for a period of 5 weeks

Haemato-biochemical parameter	Day 0	Day 7	Day 14	Day 21	Day 28
Haemoglobin	9.12 ±0.64	7.8 ±1.12	8.08 ±0.99	8.42 ±0.81	9.03 ±0.76
PCV	26.5 ±2.05	23.67 ±2.61	24.2 ±2.31	25.77 ±2.52	26.47 ±2.33
BUN	104.08 ±21.21	87.48 ±30.37	81.05 ±23.01	76.07 ±19.43	76.12 ±17.82
Serum Creatinine	8.13 ±2.03	6.9 ±1.98	6.71 ±1.92	5.62 ±1.44	5.92 ±1.65
Serum Sodium	135.48 ±3.32	135.67 ±4.76	136.2 ±2.65	138.08 ±2.2	137.15 ±2.19
Serum Potassium	4.33 ±0.34	4.19 ±0.26	4.21 ±0.35	4.44 ±0.26	4.16 ±0.22
Serum Chloride	96.07 ±3.58	102.13 ±4.35	96.25 ±1.22	102.3 ±3.48	101.73 ±2.46
Serum Phosphorus	7.52 ±0.93	6.88 ±0.86	6.29 ±0.77	6.31 ±0.45	5.97 ±0.35

Conclusions

Conservative management of CKD in dogs mainly includes a combination of treating the specific clinical signs and complication associated with CKD, maintaining adequate hydration and nutrition and slowing the loss of kidney function, which can be attained by dietary modifications, managing gastrointestinal signs, maintaining hydration, correcting hyperphosphatemia, metabolic acidosis and hypokalemia, management of anemia and proteinuria, and correction of arterial hypertension.

Treatment options for dogs with CKD should include therapy targeted towards treatable primary kidney disease (e.g. Pyelonephritis, nephroliths, etc) and conservative medical management of disease.

Dogs with IRIS Stage 3 and 4 CKD tend to have progressive loss of renal function, with a typical lifespan of a few months to 1-2 years, however quality of life may be prolonged and length may be enhanced markedly with appropriate therapy. Factors that point to poorer prognosis, such as hypertension and hyperphosphatemia, may also be managed medically to some degree.

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