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Abhishek Kumar

Assistant Professor

Department of Veterinary
Medicine, Ranchi Veterinary
College, Kanke, Ranchi,
Jharkhand, India

Praveen Kumar

Assistant Professor

Department of Veterinary
Medicine, Ranchi Veterinary
College, Kanke, Ranchi,
Jharkhand, India

Effectiveness of conservative therapy in chronic renal failure in dogs

Abhishek Kumar and Praveen Kumar

Abstract

Incidence of chronic renal failure was higher in middle to old aged dogs as compared to young dogs. High level of Azotemia and hyper-phosphatemia combined with history and physical examination were helpful in diagnosis of CRF. Conservative medical management formulated to maintain fluid, electrolyte, acid-base, nutrient & vitamins balance, control of hyper-phosphatemia and uremic gastropathy were successfully to minimize the clinical and patho-physiological consequences of reduced renal function due to CRF.

Keywords: canine, CRF, conservative treatment

Introduction

Chronic renal failure is a common renal disease in dogs characterized by severe renal damage [1]. It may be acquired, congenital or endocrine disorder caused due to infections, autoimmune diseases and toxic chemical substance [2]. Early diagnosis of renal disease through urine and blood tests enable us timely application of therapeutic interventions that may slow down disease progression [3]. The therapeutic procedure that reduces proteinuria may prolong the survival of dogs suffering from CRF [4]. Conservative therapy must be initiated as early as possible to control symptoms, minimize complications and slow progressions of disease. The present work was aimed at examination of various factors like incidence, physical and clinical findings, diagnostic features and therapy and its efficacy associated with chronic renal failure.

Materials and Methods

The study was conducted on 18 dogs diagnosed with CRF in the Deptt. of Vet. Medicine, RVC during 2018-19. The data pertaining to age, breed, sex, onset of symptoms, history pertaining to vomiting, urination and previous medication were collected. The cases were thoroughly examined for physical parameters (temp., pulse, heart and respiration rate) and clinical signs (anorexia, polyuria, polydipsia and vomiting), oral cavity for ulceration and visible mucous membrane were also examined. Blood samples were collected for hematological (Hb%, TEC, TLC & DLC) as well as serum biochemical tests (BUN, Creatinine, phosphorus, & Glucose) through standard technique (using automatic blood analyzer and biochemical analyzer) twice weekly. Urine samples were examined for specific gravity and presence of protein. All the dogs were treated with Ringer's lactate soln. (RL) and 5% DNS through i/v, Torsemide @ 0.2mg/kg b.wt. i/v, Renitidine @ 0.5mg/kg b.wt i/m, Cefotiofur Sod. @.5mg/kg b. wt. s/c, supported with Imferon @1ml, B- Complex with liver extract @ 2 ml I/m and Sucralfate suspension @ 5ml bid orally/day. Therapy was given until serum creatinine level becomes normal and continued 3 days thereafter. The data were analyzed by t-test [5].

Results

Incidence of CRF revealed 22.23% in 1 – 5 years, 33.34% in 6 – 10 years and 44.45% in 11 – 15 years old dogs. The breed wise incidence revealed highest in German shepherd (27.78%), and Labradors (27.78%) followed by Pomeranians (16.67%), Cross-breed (16.67%) and Mongrels (11.12%). The males (61.12%) were more prone to CRF than the female (38.89%) dogs. On the basis of history Vomiting, Anorexia, Polydipsia, Polyuria, Melena, Oral ulcers and Convulsions were typical findings of CRF disease noted in 98.45%, 88.89%, 66.67%, 55.56%, 33.34%, 27.78%, and 11.12% dogs respectively. A non-significant variation in Hb%, TEC, TLC & DLC of surviving dogs before and after treatment was observed (Table - 1).

Corresponding Author:

Abhishek Kumar

Department of Veterinary
Medicine, Ranchi Veterinary
College, Ranchi, Jharkhand,
India

However, these dogs revealed normocytic normochromic anemia.

Table 1: Haemato-biochemical parameters before and after treatment

Parameters	Before treatment	After treatment	Died
TEC ($\times 10^6/\mu\text{l}$)	4.84 \pm 0.36	5.26 \pm 0.18 NS	
Hb%(g/dl)	9.68 \pm 1.26	10.12 \pm 0.98 NS	
TLC(μl)	17128 \pm 1325	17096 \pm 894 NS	
DLC (%):			
Neutrophil	70.68 \pm 0.60	70.34 \pm 0.88 NS	
Lymphocyte	20.60 \pm 0.86	20.00 \pm 0.47 NS	
Eosinophil	2.63 \pm 0.35	3.01 \pm 0.28 NS	
Monocyte	5.87 \pm 0.36	6.28 \pm 0.62 NS	
BUN(mg/dl)	55.86 \pm 5.83	20.42 \pm 1.44*	98.62 \pm 10.34
Creatinine (mg/dl)	4.02 \pm 0.23	1.00 \pm 0.68*	10.24 \pm 1.47
Phosphorus (mg/dl)	8.24 \pm 1.18	6.05 \pm 0.56*	12.72 \pm 1.82
Glucose (mg/dl)	77.82 \pm 7.98	86.32 \pm 2.13*	66.92 \pm 3.24
Sp. Gravity	1.0143 \pm 0.0012	1.0298 \pm 0.0014	1.0064 \pm 0.0016

The level of BUN, creatinine and phosphorus were significantly reduced in surviving dogs after the treatment (Table-1). The mean serum glucose level in surviving dogs was 86.32 \pm 2.13 mg/dl after treatment as compared to 66.92 \pm 3.24 mg/dl in dogs that died. The mean specific gravity of urine was 1.0143 \pm 0.0012 and 1.0298 \pm 0.0014 before and after treatment respectively and dog that died following therapy was specific gravity of 1.0064 \pm 0.0016. Clinical improvement in appetite and cessation of vomiting was observed between 3 – 7 days in surviving dogs. Out of 18 dogs treated, 10 completely recovered and 8 died during therapy. The levels of BUN decreases faster as compared to serum creatinine levels following therapy in surviving dogs as BUN levels reached to normal in 3 to 7 days whereas serum creatinine in 7 – 10 days.

Discussion

CRF is an uncommon disease in middle to old aged dogs showing typical signs of vomiting, anorexia, polydipsia and polyuria [6]. Oral ulceration may occur due to breakdown of urea in saliva to ammonia by the oral bacteria [7].

The increase in Creatinine and BUN levels in present findings is in respect with the reports of other researchers [6, 8]. The mean BUN concentration in dogs that died during treatment was 98.62 \pm 10.34 mg/dl such high levels of BUN following treatment are consistent with the findings of other workers [9, 10].

The increased mean of serum phosphorus levels (8.24 \pm 1.18 mg/dl) before treatment was found declined significantly following therapy and dog that died were remains to 12.72 \pm 1.82mg/dl such hyperphosphatemia are consistent with the results of other workers [11,12]

Hence, conservative medical management formulated to maintain fluid, electrolyte, acid- base, nutrient & vitamins balance, control of hyper-phosphatemia and uremic gastropathy were successful to minimize the clinical and patho-physiological consequences of reduced renal function due to CRF.

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