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Clinical And Nephrosonographic Findings In Canine Chronic Renal Failure: A Prospective Study

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Abstract: *In this study, the clinical and ultrasonographic findings of chronic renal failure (CRF) in dogs is presented with relation demographics in a total of 31 CRF dogs. The diagnosis involved clinical observations, hematology, serum biochemical profile, urinalysis and ultrasonography. The respective findings were compared with 10 healthy control dogs. CRF with male predominance especially in 8 to 12 years dogs and in spitz breed was observed. The predominant signs in CRF dogs included anorexia, vomition, dullness, weight loss, oral ulcers, polyuria, and polydipsia, pallor of mucosa, hypertension recumbency and blindness. Blood picture revealed anemia with mild neutrophilic leukocytosis. Serum urea nitrogen, creatinine, sodium and phosphorus levels were significantly elevated whereas total protein and albumin were reduced. Urine had lower specific gravity and contained higher amounts of protein, creatinine, alkaline phosphatase and gamma glutamyl transferase enzymes. Nephrosonography revealed hyperechoic renal cortex and medulla, indistinct cortico-medullary junction and shrinkage of kidneys.*

Keywords – *Chronic renal failure, Hypertension, Nephrosonography, Dogs, Incidence, Kidney, Diagnosis*

I. Introduction

Chronic renal failure (CRF) is a common problem in aged dogs and is associated with significant morbidity and mortality. Several risk factors are attributed for CRF including old age, specific breeds, smaller body size, periodontal disease and obesity [1]. It results from a progressive and irreversible loss of functioning nephrons and the cause is often difficult to determine. Dogs with CRF exhibit polyuria, polydipsia, anorexia, vomiting, weight loss, pallor of mucous membrane, oral ulceration, halitosis and acute blindness [2]. Patient history, results of physical examination, urinalysis, hematology, serum biochemistry and nephrosonography provide a practical means of diagnosing CRF in dogs. In spite of the poor long term prognosis, patients with CRF often survive for many months to years with a good quality of life. Although no treatment can correct existing irreversible renal lesions of CRF, the clinical and biochemical consequences of reduced renal function can often be ameliorated by symptomatic and supportive therapy. Early diagnosis and a structured approach to CRF investigation and therapeutic management may slow down disease progression in dogs. Conservative medical management of CRF includes providing adequate and appropriate nutrition, correcting fluid deficit, electrolyte abnormalities and acid base imbalance, ameliorate clinical signs of CRF and providing reno-protective therapy to slow down the disease progression [3, 4]. Hemodialysis, chronic ambulatory peritoneal dialysis and renal transplantation are the advanced methods of treatment for CRF but their expense, technical difficulties and limited experience restricts their routine use in veterinary practice. Recently, herbal drugs are gaining importance both in research and clinical usage for treating human and veterinary ailments [5-14]. It is suggested that adequate nutrition combined with conservative therapy and herbal drugs might increase the survival time in dogs with CRF [15].

II. Material And Methods

2.1 Clinical cases

Thirty one clinical cases of dogs brought to the small animal clinical complex with clinical cases suggestive of chronic renal failure (CRF) were included in the study. Ten apparently healthy dogs of different breeds aged between three to five years of age were selected as control group for obtaining normal values for various parameters.

2.2 Clinical examination and diagnosis

Detailed clinical examination including monitoring of blood pressure, hematology – hemoglobin, packed cell volume, total erythrocyte count, total leucocyte count and differential count; serum biochemical parameters – blood urea nitrogen, creatinine, total protein, albumin, sodium, potassium, phosphorus and calcium; urinalysis – pH, specific gravity, creatinine, protein, alkaline phosphatase and gamma glutaryl transferase. Nephrosonography was performed to study the architecture of kidneys.

2.3 Blood pressure monitoring

Human wrist model automatic oscillometric sphygmomanometer (BPL Ltd. India) was for measuring blood pressure in dogs in sternal recumbency, the cuff was placed on the left forelimb and transducer on the medial aspect of the arm over the median artery.

2.4 Hematology and sero-biochemical profile

Three mL of whole blood collected from saphenous vein. One mL of whole blood was used for hematology and remaining blood was used for serum collection. In serum, parameters such as creatinine, urea nitrogen, total protein, sodium, potassium, calcium, phosphorus were analyzed using standard kits supplied by Span diagnostics Ltd., Surat using star 21 semi-auto biochemistry analyzer (Rapid Diagnostic Pvt. Ltd, Delhi)

2.5 Urinalysis

Cystecentesized urine was collected and urine pH, specific gravity and protein were determined using URISCAN dip sticks. Alkaline phosphatase (ALP) and gamma glutamyl transferase (GGT) were estimated from after separating sediments from urine using standard kits supplied by Accurex biomedical Pvt. Ltd, Mumbai.

2.6 Nephrosonography

Nephrosonography was performed in either dorsal or sternal recumbency using IXOS vet-ultrasound machine supplied by Esoate Pie medical, Netherlands. Probes of 3.5 MHz was used for small, 5.0 MHz for medium and 7.5 MHz for large dogs. Left kidney was imaged caudal to the greater curvature of the stomach, caudo-dorsal to the spleen, later to the aorta and left to adrenal gland at the level of L2 to L4 vertebrae. The right kidney was imaged caudal to right liver lobes, lateral to the caudal venacava and right adrenal gland at the level of L1 to L3 vertebrae [16]. The echogenicity of the identifiable lesion, as seen on the grey scale 2-D images were classified subjectively as normal, increased (hyperechoic), and decreased (hypoechoic), absent compared to normal echo pattern for canine kidney [17].

2.7 Statistical Analysis

The data for various parameters was expressed as Mean±S.E. The values for each parameter were compared to control values by student's t-test using Statistical package for social sciences (SPSS) 19.0V. The level of significance was set at $P<0.05$.

III. Results

3.1 Distribution

CRF was highest in 8 to 12 years (38.7%) and least in less than 4 years of age (12.9%). A male (58.1%) predominance over female (41.9%) was observed. Among the affected breeds, Spitz breed (54.84%) showed highest incidence followed by German shepherd (16.13%), and least in Doberman pinscher (3.23%). (Table 1).

Table 1 Demographic distribution of dogs with Chronic renal failure

Age (Y)	No. of cases	Percentage
0-4 y	4	12.9
4-8 y	8	25.8
8-12 y	12	38.7
>12 y	7	22.6
Breed		
Spitz	17	54.84
Mongrel	4	12.90
German shepherd	5	16.13
Labrador retriever	2	6.45
Great Dane	2	6.45
Doberman pinscher	1	3.23
Sex		
Male	18	58.10
Female	13	41.90

3.2 Clinical signs

In CRF dogs the predominant symptoms were anorexia (64.5%), vomition (48.38%), dullness and weight loss (45.16%) and oral ulcers (38.7%) followed by hypertension (19.35%) polyuria and polydipsia (16.12%), recumbency (12.9%) and blindness (9.67%). (Table 2)

Table 2 Clinical findings in dogs suffering from chronic renal failure

Symptom	No. of cases	Percentage
Anorexia	20	64.51
Vomitions	15	48.38
Weight loss	14	45.16
Dullness	14	45.16
Oral ulcers	12	38.7
Polyuria/Polydipsia	6	19.35
Hypertension (>150mm Hg)	6	19.35
Melena	5	16.12
Pallor of mucous membrane	5	16.12
Recumbency	4	12.9
Blindness	3	9.67

3.3 Blood pressure: The mean systolic and diastolic arterial pressure of CRF dogs (134.81±1.77 and 80.61±1.75) was significantly (P<0.05) higher than control dogs (121.1±1.80 and 71.1±1.33) respectively. (Table 3)

3.4 Hemogram: CRF dogs had a significantly (P<0.05) lower erythrocyte count (4.66±0.19 vs 7.58±0.32) and an associated decrease in packed cell volume (32.58±0.73 vs 43.1±1.72) and hemoglobin concentration (11.87±0.32 vs 15.28±0.39) compared to control dogs. However, no significant difference between CRF and control dogs was observed in total leucocyte count and differential count. (Table 3)

Table 3 Clinical findings in chronic renal failure

Parameter	Control dogs	Chronic renal failure dogs
Systolic pressure (mm Hg)	121.10±1.80	134.81±1.77*
Diastolic pressure (mm Hg)	71.10±1.33	80.61±1.75*
Hemoglobin (g/dL)	15.28±0.39	11.87±0.32**
Packed cell volume (%)	43.10±1.72	32.58±0.73
Total erythrocyte count (10^6/mm^3)	7.58±0.32	4.66±0.19**
Total leucocyte count	9610.00±331.00	13179.00±1870.00
Neutrophils (per mm^3)	6780.00±2.22	7332.30±1.62
Lymphocytes (per mm^3)	2240.00±2.70	2183.90±1.65
Monocytes (per mm^3)	890.00±1.26	574.20±0.79
Eosinophils (per $mm^{3)}$	190.00±0.43	134.40±0.18

Values are mean± standard error; Student's t-test using SPSS 19.0 V
*P<0.05; **P<0.01

3.5 Serum biochemical profile

In CRF dogs in comparison with control dogs showed significant elevated creatinine (4.91±0.49 vs 0.45±0.05), urea nitrogen (183.16±19.43 vs 19.00±2.24), sodium (167.32±3.00 vs 145.22±4.19), phosphorus (6.62±0.18 vs 3.88±0.23) and a significant decrease (P<0.05) was observed in total protein (6.39±0.19 vs 7.26±0.31) and albumin (2.41±0.15 vs 3.54±0.16) concentrations. (Table 4)

3.6 Urinalysis

There was a significant decrease in specific gravity of urine (1.020 vs 1.036) and increased levels of urinary protein (217.42±44.95 vs 6.94±0.55), alkaline phosphatase (9.04±0.37 vs 1.62±0.09) and gamma-glutaryl transferase (8.13±0.24 vs 1.55±0.09) compared to control dogs. (Table 5)

3.7 Nephrosonography

Ultrasonography revealed that the kidneys in CRF dogs was predominantly indistinct or absent cortico-medullary junction (78% dogs) followed by altered renal architecture (65%), hyperechoic cortex (25%) and shrinkage of kidneys (16%).

Table 4 Serum biochemical profile in dogs suffering from chronic renal failure

Parameter	Control dogs	Chronic renal failure dogs
Blood urea nitrogen (mg/dL)	19.00±2.24	183.16±19.43**
Creatinine (mg/dL)	0.45±0.05	4.91±0.49**
Total Protein (g %)	7.26±0.31	6.39±0.19*
Albumin (g %)	3.54±0.16	2.41±0.15*
Sodium (mEq/L)	145.22±4.19	167.32±3.00**
Potassium (mEq/L)	4.61±0.16	4.03±0.53
Phosphorus (mg/dL)	3.88±0.23	6.62±0.18**
Calcium (mg/dL)	9.75±0.49	9.32±0.16

Values are mean± standard error; Student's t-test using SPSS 19.0 V
*P<0.05; **P<0.01

Table 5 Urinalysis of dogs suffering from chronic renal failure

Parameter	Control dogs	Chronic renal failure dogs
pH	6.67±0.17	6.66±0.05
Specific gravity	1.036±0.02	1.020±0.001**
Urinary creatinine	10.14±0.89	217.42±21.20**
Urinary protein (mg %)	6.94±0.55	217.42±44.95**
Urinary protein/Creatinine ratio (UP/C)	0.68±0.09	2.29±0.25**
Urinary Alkaline Phosphatase (mmol/ mg Creatinine)	1.62±0.09	9.04±0.37**
Urinary Gamma-glutaryl transferase (mmol/ mg Creatinine)	1.55±0.09	8.13±0.24**

Values are mean± standard error; Student's t-test using SPSS 19.0 V
*P<0.05; **P<0.01

IV. Discussion

Chronic renal failure (CRF) is an important clinical condition in dogs, which results from reduced renal function leading to accumulation of substances normally excreted by kidneys. As clinical signs of CRF are non-specific, many cases go unnoticed in veterinary practice. Investigation of age-wise distribution of CRF revealed that old dogs (8-12y) are more commonly affected [3]. High incidence of CRF in Spitz breed of dogs observed in this study is more likely due popular choice of this breed as a pet. CRF also showed male predominance [18].

Clinical signs such as anorexia, vomition, weight loss, oral ulcers, polydipsia, and polyuria, pallor of mucosa, recumbency and blindness are commonly observed in CRF [2]. Anorexia and vomition is caused due to the stimulation of chemoreceptor trigger zone (CTZ) by uremic toxins [19]. Weight loss and dullness are consequences of inadequate calorie intake, insulin resistance and combined catabolic effects of uremia and intestinal malabsorption [20]. Increased loss of water and important substances results in polydipsia and electrolyte imbalances.

In this study, a systolic and diastolic pressure measured by indirect oscillometric method showed a significant increase in CRF dogs compared to control. These findings are in agreement with earlier observations [22]. CRF in dogs may precipitate hypertension, which worsens existing renal pathology and contributes to disease progression [23]. The blood picture revealed a significant decrease in hemoglobin, packed cell volume and total erythrocyte count, which concur with earlier workers [24]. Out of 31 dogs presented to the study, 11 dogs were found to be anemic. Cowgill et al. [25] listed the causes of anemia in CRF as reduced renal production of erythropoietin, reduced red blood cell survival, gastrointestinal bleeding and uremic inhibitors of erythropoiesis, bone marrow fibrosis and nutritional deficiencies. Although the leucocyte and differential count were normal in CRF dogs, leukocytosis is a common finding in CRF, which is an indicator for progression of kidney disease [26].

Serum biochemical profile showed significantly elevated urea nitrogen and creatinine values in CRF dogs, which is in agreement with earlier observations [24, 27]. Serum creatinine levels are commonly used to measure kidney dysfunction and is the basis for the international renal interest society (IRIS) staging system [29]. Raised urea nitrogen and creatinine levels in CRF dogs results from compromised kidney function leading to retention of nitrogenous substances [15, 24]. Earlier works reported that rise in serum urea and creatinine occurs when there is severe renal impairment [28, 30]. In addition, GIT hemorrhages are also known to contribute to increased urea in blood as a consequence of increased GI absorption of nitrogenous compounds [31]. A significant decrease in total protein and serum albumin values in CRF dogs can be attributed to increased filtration of albumin through glomeruli, owing to its molecular size [32]. Serum potassium and calcium were within normal range in CRF dogs where as a significant increase in serum phosphorus and sodium was observed. These findings are in agreement with earlier workers [24, 27]. The rise in serum phosphorus and sodium could be a result of declining glomerular filtration rate in CRF resulting hyperphosphatemia and hypernatremia [25].

In urinalysis, a significant decrease in urine specific gravity and highly significant increase in urine protein and creatinine and urinary enzymes (ALP and GGT) excretion in CRF in dogs. The decrease in urine specific gravity in CRF dogs is a result of decreased concentrating ability of kidneys. Proteinuria and increased UP/C ratio, urinary enzymes (ALP and GGT) in CRF dogs could be attributed to the glomerular damage [3, 33].

In normal control dogs the sonographic architecture containing a mixture of hyperechoic, hypoechoic and anechoic patterns. The medulla was hypoechoic, round in appearance with well-defined cortico-medullar junctions. At the center the hyperechoic renal pelvis was noticed. The ultrasonographic changes observed in CRF dogs were consistent with the findings of earlier workers [34]. Walter et al. [17] reported increased overall echogenicity and reduced corticomedullary definition in dogs with chronic inflammatory and end stage renal disease. End stage renal disease kidneys are typically small, irregular and diffusely echogenic with poor visualization of cortico-medullar junction and hyperechoic medulla. Rosenfield [35] reported hyperechoic cortex results from deposition of calcium in renal cortex.

V. Conclusion

Chronic renal failure is common progressive disease of kidneys and occurs mainly in old dogs with male predominance. This condition is characterized by hypertension, anemia, elevated serum urea nitrogen, creatinine and elevated urinary protein and urinary enzymes. Nephrosonography indicated hyperechoic medullary junction, altered renal architecture, hyperechoic cortex and shrinkage of kidneys. The survival of dogs can be improved by using conservative therapy combined with herbal drugs.

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