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Chronic Kidney Disease in a Dog: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. Authors MNUN and SZ collected blood and urine samples, conducted biochemical analyses, and performed X-rays and ultrasounds. Author NH formulated the renal diet and drafted as well as reviewed the manuscript. Author MMHB was responsible for patient follow-up and conducted an X-ray after the patient's recovery. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

This report describes a 10-year-old Labrador Retriever dog who was found to have chronic kidney disease (CKD). Classic CKD signs, such as weight loss, increased thirst, decreased appetite and frequent urine were observed in the patient. Tests in the lab revealed proteinuria coupled with increased blood urea nitrogen and creatinine levels. The renal cortex displayed structural changes measuring 7.5 mm on ultrasound. Radiographic evaluation revealed an enlarged heart, with the vertebral heart score (VHS) elevated at 12.5v in the right lateral view, exceeding the reference range of 10.2–11.4v. Additionally, cardiac troponin-I levels were elevated, further supporting evidence of cardiac involvement. A comprehensive treatment plan was initiated. Medication to control blood pressure, supportive care for secondary problems, and a renal diet to reduce protein

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and phosphorus consumption. To evaluate the effectiveness of the treatment and make the required modifications, routine monitoring of kidney function and general health was essential. Two months later, all the biochemical parameters improved and the renal cortex returned to normal, measuring 6.3 mm. Additionally, cardiac troponin-I levels and heart size, as indicated by the VHS score, normalized to 10.5v, reflecting the positive response to long-term therapy for chronic kidney disease (CKD) and its associated cardiac complications. This case underscores the critical need for early diagnosis and appropriate treatment in dogs with CKD to enhance quality of life and extend lifespan.

Keywords: Chronic Kidney disease; creatinine; dog; troponin.

1. INTRODUCTION

In canines, chronic kidney disease (CKD) is a prevalent and serious condition marked by a progressive and irreversible loss of kidney function. Weight loss, vomiting, changes in appetite, and increased thirst and urination (polydipsia/polyuria) are some of the minor symptoms that may be present in the early stages of CKD (Bartges, 2012). CKD is defined by structural and functional abnormalities in one or both kidneys lasting at least three months. In affected dogs and cats, CKD typically results in a permanent reduction in functional nephrons, which are the essential filtering units of the kidney (Polzin, 2011). The prevalence of CKD in dogs is estimated to be 5.8% of the veterinary caseload (Bartlett et al., 2010) with geriatric dogs being most commonly affected. Approximately 15% of dogs over 10 years of age have been reported to show structural and functional changes in the kidneys (Polzin, 2011). While CKD is often associated with older animals, it can occur in dogs and cats of all ages, with incidence rates in the general population estimated at 0.50–1.50% in dogs and 1–3% in cats (Elliott & Grauer 2007). CKD can manifest similarly in dogs and humans, leading to progressive renal failure, uremic crisis and potentially death (Hasegawa et al., 2017). CKD can arise from a variety of causes including congenital disorders like polycystic kidney disease or renal dysplasia, glomerulonephritis secondary to neoplasia or infection or idiopathic causes (Lefebvre, 2011). Diagnosis in dogs is typically based on elevated serum creatinine, blood urea nitrogen (BUN) and phosphorus levels, with up to 75% of kidney function potentially lost before azotemia becomes detectable (Polzin, 2011). Chronic kidney disease (CKD) often leads to secondary hypertension, which is a major contributor to cardiac complications such as dilated cardiomyopathy and cardiomegaly in dogs. Other endocrine disorders, such as hyperthyroidism, hyperadrenocorticism, and diabetes mellitus, also play a role in the development of this condition (Ware, 2007). Cardiomyopathy is characterized by the enlargement of the ventricular chambers of the heart and impaired systolic function (Simpson et al., 2015). It is frequently associated with chronic secondary hypertension, which can arise as a consequence of chronic kidney disease CKD (Sarnak, 2008). Cardiac biomarker, such as cardiac troponin-I (cTnI) is important in assessing cardiovascular damage in dogs (Sharkey et al., 2009). Besides amino-terminal pro-B-type natriuretic peptide (NT-proBNP) is also an important biomarker for cardiac disease (Schmidt et al., 2009). Elevated levels of these biomarkers have been documented in dogs with azotemia, though it is unclear whether they indicate cardiorenal syndrome (CvRDk) or result from decreased glomerular filtration rate (GFR) (Boswood et al., 2008).

Once CKD is diagnosed, the focus shifts to management and treatment, with dietary phosphate restriction and specially formulated renal diets being among the most effective strategies (Elliott, 2006). Management aims to slow disease progression and improve the quality of life through fluid therapy, dietary modification and pharmacological intervention. Regular monitoring and a comprehensive understanding of CKD's impact on renal function are essential for optimizing outcomes in affected dogs. This report will explore the clinical presentation, diagnostic approaches, and management strategies for chronic kidney disease (CKD) and its associated cardiac complications in dogs, highlighting the significance of early diagnosis and long-term care.

2. CASE PRESENTATION

2.1 Clinical and Physical Examination

In Purbachal, Dhaka, Bangladesh, a 10-year-old Labrador Retriever male local dog was brought

to the Teaching and Training Pet Hospital and Research Center after exhibiting signs of dehydration, decreased appetite and noticeable weight loss for 3 months. On clinical examination, polyuria and polydipsia were found along with a sunken aspect of the eyes. The dog was found to have a lower-than-normal body temperature of 97.3ºF, heart rate (135 beats/min), respiration rate (40/min). Blood pressure measurements were obtained multiple times using oscillometry. The average systolic pressure was 130 mmHg, while the diastolic pressure was 85 mmHg. However, the patient had a prior history of hypertension. Chronic kidney disease (CKD) was first suspected based on these clinical indicators.

2.2 Sample Collection and Laboratory Investigation

Blood samples were collected under fasting conditions prior to treatment. For the biochemical analysis, two different kinds of vacutainers were used to collect the blood samples: one with an anticoagulant (K3-EDTA) and one without an anticoagulant. Blood urea nitrogen (BUN), blood glucose, total protein (TP), serum glutamic-pyruvic transaminase (SGPT), serum glutamic-oxaloacetic transaminase (SGOT), serum creatinine, calcium, phosphorus, potassium, globulin, albumin, and troponin-I were among the parameters that were assessed after serum from blood samples without an anticoagulant was separated by centrifugation at 3000 rpm for 10 minutes. Roche Diagnostics GmbH's Cobas® E411 analyzer series was used for all biochemical testing.

2.3 Ultrasonography (USG)

Ultrasonography (USG) of the lower ventral abdomen was used to further confirm CKD. To get ready for the procedure, a disposable blade was used to shave the ventral abdomen of the dog. The USG probe was used to detect the cortex of both kidneys in the lower ventral abdomen following the proper amount of constraint. Using a 15A probe, the ultrasonography was conducted at 4.0 MHz to acquire comprehensive kidney pictures for evaluation.

2.4 Radiography

A right lateral view chest radiography was obtained to assess the vertebral heart score (VHS).

3. DISCUSSION

3.1 Final Diagnosis

The dog presented with polyuria, polydipsia, anorexia, and weight loss, which are indicative of early-stage chronic kidney disease (CKD). Additionally, the dog exhibited signs of weakness and anemia, both of which are also characteristic of CKD. Biochemical analysis revealed total protein (TP) at 11.8 g/dL and globulin at 9.30 g/dL, both higher than the reference values (Cornell University College of Veterinary Medicine n.d.).

Creatinine, a key marker of kidney function, was elevated at 5.7 mg/dL, confirming CKD as the definitive diagnosis (Polzin, 2011). The blood urea nitrogen (BUN) level was also high, at 75 mg/dL. Urinalysis revealed normal pH and specific gravity levels, but there was a significant presence of protein, reinforcing the diagnosis of kidney disease. All the hematological parameters remained within the normal range. The complete hematological and biochemical findings are summarized in Table 1. Proteinuria is a well-established independent risk factor for the progression of CKD in dogs and cats (Syme et al., 2006). The phosphorus level was found to be 13.2 mg/dL, which exceeded the reference range (2.7-5.4 mg/dl). Phosphorus is typically filtered by the kidneys, and elevated levels in the blood are common when kidney function declines (Polzin, 2011). Ultrasound findings revealed increased cortical thickness in the kidneys which is 7.5 mm (Fig. 2), which was suggestive of chronic kidney disease.

The elevated cardiac-specific troponin I (cTnI) was 0.39 ng/ml in serum concentration, exceeding the normal reference value 0.05-0.24 ng/ml (Spratt et al., 2005). X-ray imaging (right lateral view) revealed cardiac enlargement, with a vertebral heart score (VHS) of 12.5v, exceeding the reference range of 10.2–11.4v (Fig. 1). The clinical significance of this relationship in canines remains unclear, some have proposed the term cardiovascular-renal axis disorders (CvRD) to describe such interactions in companion animals (Pouchelon et al., 2015). CvRD secondary to kidney disease (CvRDk) has been suggested to involve harmful interactions between the cardiovascular and renal systems, although direct evidence in dogs is limited. However, factors such as hyperkalemia, anemia, hypertension and fluid imbalances (hypervolemia

or hypovolemia) have been documented. Impaired renal excretion of toxic substances may also negatively impact the cardiovascular system (Pelander, 2018).

Table 1. Hematological and biochemical parameters of blood and urine in dog affected by chronic kidney disease

Name of test	Result	Reference value
Serum analysis		
Calcium(mg/dL)	9.30	9.40-11.10
Phosphorus(mg/dL)	13.20	2.70-5.40
Potassium(mEq/L)	4.70	4.10-5.40
Glucose(mg/dL)	110.3	68.0-104.0
Total protein(g/dL)	11.8	5.50-7.20
Albumin(g/dL)	2.50	3.20-4.10
Globulin(g/dL)	9.30	1.90-3.70
SGPT(U/L)	65.00	17.00-95.00
SGOT(U/L)	80.00	18.00-56.00
ALP(U/L)	12.00	7.00-115.00
BUN (mg/dL)	75.00	9.00-26.00
Ceatinine(mg/dL)	5.70	0.60-1.40
Cardiac Troponin I (ng/ml)	0.39	0.05-0.24
Hematological analysis		
Red Blood Cells (million/µL)	6.2	5.5-8.5
White Blood Cells (k/µl)	11.5	8-17
Neutrophils (k/µl)	10.4	3-11.8
Lymphocytes (k/µl)	0.5	1-4.8
Monocytes (k/µl)	0.42	0.1-2
Eosinophils (k/µl)	0.20	0-1.3
Basophils (k/µl)	0.01	0-0.5
Packed cell volume (%)	41	37-50
Hemoglobin (gm/dl)	11.8	12-18
Urine analysis		
pH	6.5	6.0-7.0
Specific gravity	1.012	1.001-1.065
Glucose (mg/dl)	200	180-220
Protein(gm/dl)	4.0	0.5-1.0

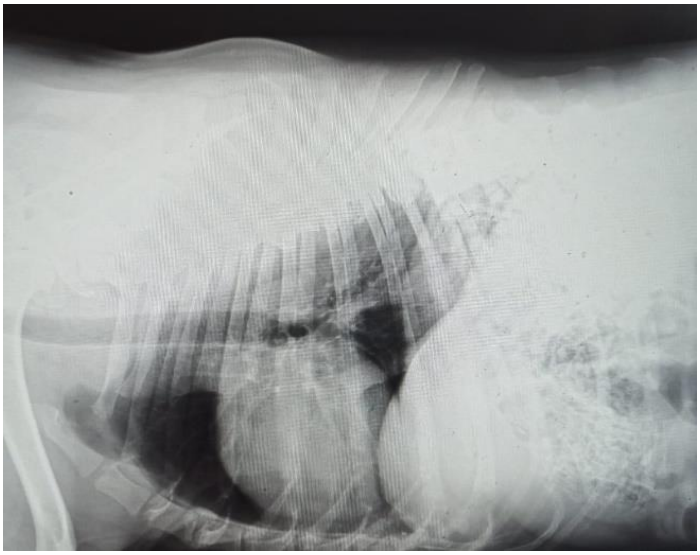

Fig. 1. X-ray of Dog (Enlarged Heart) Veterbral Heart Score (12.5v)

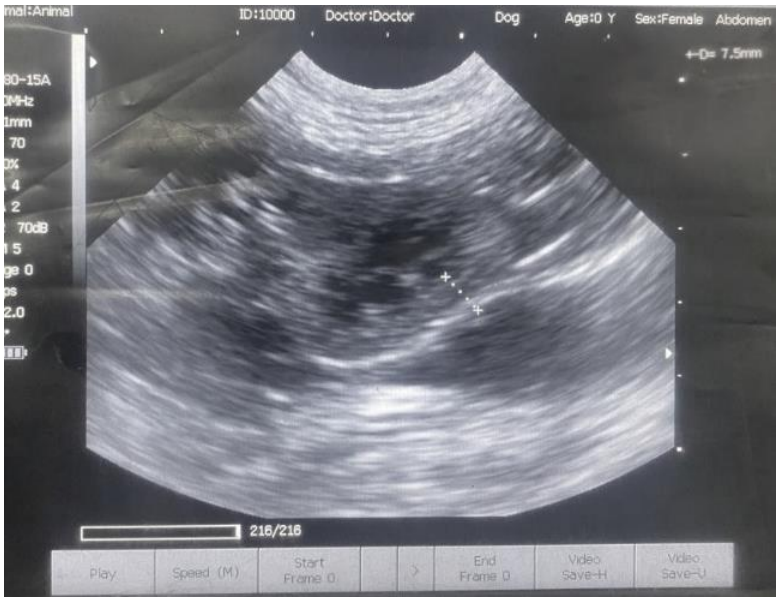

Fig. 2. Ultrasound of Dog (Increased Renal Cortex Size) 7.5mm

3.2 Treatment and Outcome

According to the International Renal Interest Society (IRIS) guidelines (Table 2), the dog in this case was classified as stage 4 CKD. In stage 1, non-azotemic conditions are characterized by renal abnormalities other than azotemia, such as abnormal findings on renal imaging or palpation or a progressively increasing creatinine level. Stage 2 involves mild renal azotemia, where clinical signs are typically mild or absent. Stage 3 is marked by moderate renal azotemia, often accompanied by systemic clinical signs. In stage 4, severe renal azotemia is present, with systemic clinical signs usually evident (Gunawan et al., 2023).

Effective CKD management requires appropriate treatment combined with a renal diet. For our 45 kg dog, we provide a balanced renal diet consisting of 121 grams of cooked, roasted chicken meat, 59 grams (or 13 teaspoons) of canola oil, and 10.90 grams (approximately 10 mL) of Nordic Naturals Omega-3 Pet Liquid. Additionally, we include 217 grams (around 1 3/8 cups) of cooked rice without salt, 0.38 grams (about 1/16 teaspoon) of salt, and 7.8 grams of Wholistic Pet Organics multivitamins. Phosphorus restriction was introduced due to the elevated phosphorus levels and fluid therapy was administered to correct electrolyte imbalances and restore normal phosphorus and potassium levels. Common phosphate-binding agents for dogs with CKD include aluminum hydroxide, oxide and carbonate salts, though there is a risk of aluminum toxicity when used at high doses in advanced CKD (Segev et al., 2008). To mitigate this risk, alternative non-aluminum-based phosphate binders, such as calcium carbonate (90 mg/kg) and calcium acetate (60mg/kg), were considered. Omega-3 polyunsaturated fatty acids (PUFA) (1mg/kg)were also included in the dog's diet, which has been shown to improve renal function, reduce proteinuria and lower cholesterol levels in CKD dogs (Brown et al., 1998). For hypertension management, Amlodipine, a calcium channel blocker, was administered at a dosage of 0.2–0.4 mg/kg once daily, in combination with the ACE inhibitor benazepril hydrochloride (0.25–0.5 mg/kg orally once daily), demonstrating effective results. Amlodipine is often the first-choice drug for treating hypertension in cats, though clinical data on its efficacy in hypertensive dogs are limited. For improvement of enlarged heart pimobendan (Positive Inotrope & Vasodilator) 0.25 to 0.3 mg/kg orally twice a day was used. In this case, the dog showed significant improvement following treatment protocols suggested by (Polzin, 2011), which included appetite stimulants, phosphate binders, and a renal diet. Studies have shown that renal diets significantly reduce the risk of uremic crises by approximately 75% in dogs with CKD compared to those on regular adult maintenance diets. Renal diets also help reduce the severity of proteinuria in dogs with proteinuric kidney disease (Lees et al., 2005). During the two-month follow-up period, no complications were reported, and the dog was found to be in good health, with all biochemical

parameters returning to normal ranges (Table 3) and renal cortex diameter returns to normal at 6.3mm (Fig. 3) and heart size become normal (Fig. 4). The results indicate a positive response to the treatment.

Table 2. Different stages of CKD (According to International Renal Interest Society (IRIS) guidelines)

Stages	Creatinine level (mg/dL)
Stage 1	<1.4
Stage 2	1.4-2.8
Stage 3	2.9-5.0
Stage 4	>5

Table 3. Biochemical parameters of blood and urine in dog affected from chronic kidney disease

Name of test	Result	Reference value
Serum analysis		
Calcium(mg/dL)	9.70	9.40-11.10
Phosphorus(mg/dL)	5.60	2.70-5.40
Potassium(mEq/L)	4.80	4.10-5.40
Glucose(mg/dL)	109.00	68.0-104.0
Total protein(g/dL)	7.80	5.50-7.20
Albumin(g/dL)	2.90	3.20-4.10
Globulin(g/dL)	4.30	1.90-3.70
SGPT(U/L)	70.00	17.00-95.00
SGOT(U/L)	49.00	18.00-56.00
ALP(U/L)	21.00	7.00-115.00
BUN (mg/dL)	29.00	9.00-26.00
Ceatinine(mg/dL)	1.50	0.60-1.40
Cardiac Troponin I (ng/ml)	0.17	0.05-0.24
Urine analysis		
pH	6.4	6.0-7.0
Specific gravity	1.014	1.001-1.065
Glucose (mg/dl)	187.00	180.0-220.0
Protein(gm/dl)	1.30	0.50-1.00

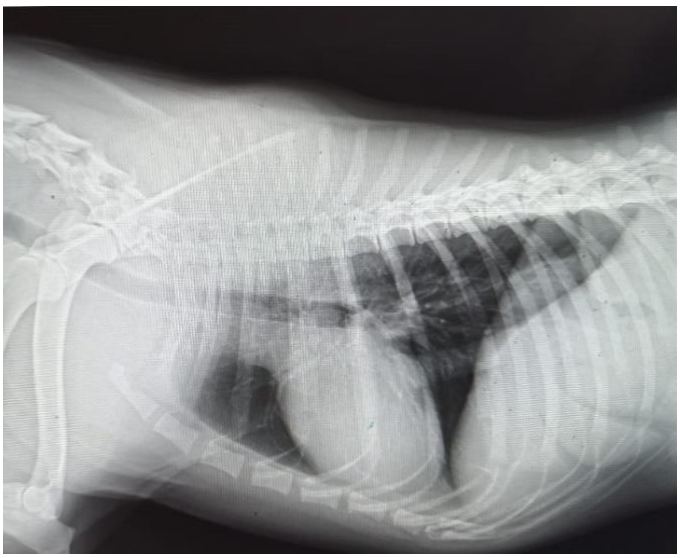

Fig. 3. X-ray of Dog (Heart Size Returns to Normal) Veterbral Heart Score (10.5v)

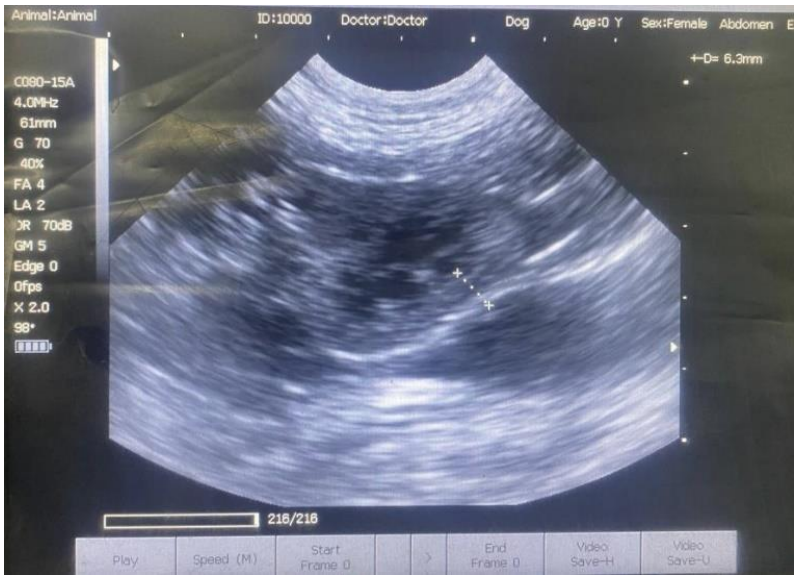

Fig. 4. Ultrasound of Dog (Renal Cortex Size Returens to Normal) 6.3 mm

4. CONCLUSION

Chronic Kidney Disease (CKD) is a prevalent and progressive condition in dogs, particularly affecting older animals but cardiac involvement is not common. This report highlights the importance of early diagnosis and comprehensive management in improving the prognosis of CKD, especially when its associated with cardiac complications. Timely intervention through dietary modifications, appropriate pharmacological treatments, and regular monitoring of biochemical markers can significantly slow the progression of the disease and enhance the quality of life for affected animals. The successful management of this case underscores the value of a multidisciplinary approach in addressing the complex interplay between kidney and cardiovascular health in canine patients. Further studies and continuous improvements in treatment protocols will be vital for optimizing outcomes in dogs with CKD.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

ETHICAL APPROVAL

Animal Ethic committee approval has been collected and preserved by the author(s).

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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