Case report

Chronic Kidney Disease with Cardiomyopathy in a Dog: A Case Report

Abstract

This report describes a 10-year-old dog who was found to have chronic kidney disease (CKD) and dialated cardiomyopathy. 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 showed 7.5 mm of structural changes on ultrasound, and the X-ray showed enlarged heart and high cardiac troponin-I indicative of dilated cardiomyopathy. A comprehensive treatment plan was initiated. Medication to control blood pressure, supportive care for secondary problems, and a renal diet to reduce protein 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, ~~the dog's~~ all the biochemical parameters, and renal cortex returned to normal at 6.5 mm, along with the troponin I value and heart size, indicating the significance of long-term therapy of chronic kidney disease (CKD) and associated consequences. This case underscores the critical need for early diagnosis and appropriate treatment in dogs with CKD and cardiomyopathy to enhance quality of life and extend lifespan.

Keywords: Kidney disease, cardiomyopathy, creatinine, dog, troponin.

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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 [1]. 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 [2]. The prevalence of CKD in dogs is estimated to be 5.8% of the veterinary caseload [3] 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 [2]. 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 0.5–1.5% in dogs and 1–3% in cats [4]. CKD can manifest similarly in dogs and humans, leading to progressive renal failure, uremic crisis, and potentially death [5]. 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 [6]. 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 [2]. CKD often leads to secondary hypertension, which is one of the major contributors to dilated cardiomyopathy in dogs. Other endocrine disorders, such as hyperthyroidism, hyperadrenocorticism, and diabetes mellitus, also play a role in the

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development of this condition [7]. Dilated cardiomyopathy (DCM) is characterized by the enlargement of the heart's ventricular chambers of the heart and impaired systolic function [8]. It is frequently associated with chronic secondary hypertension, which can arise as a consequence of chronic kidney disease CKD [9]. Cardiac biomarker, such as cardiac troponin-I (cTnI) is important in assessing cardiovascular damage in dogs [10]. Besides amino-terminal pro-B-type natriuretic peptide (NT-proBNP) is also important biomarker for cardiac disease [11]. 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) [12].

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 [13]. 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 managemental strategies associated with CKD and cardiomyopathy in dogs, underscoring the importance of early diagnosis and long-term care.

2. Case Presentation

2.1 Clinical and Physical Examination

In Purbachal, Dhaka, Bangladesh, a 10-year-old male local dog was taken to the Teaching and Training Pet Hospital and Research Center after exhibiting signs of dehydration, decreased appetite, and noticeable weight loss. 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. Chronic kidney disease (CKD) was first suspected based on these clinical indicators.

2.2 Sample Collection and Laboratory Investigation

For the analysis of biochemistry two different kinds of vacutainers were used to collect the blood samples: one with an anticoagulant (K3-EDTA) and one without of 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 dog's 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 lateral and ventro-dorsal chest radiography was performed to confirm the diagnosis of cardiomyopathy further.

3. Discussions

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 9.3 g/dL, both higher than the reference values [14]. Creatinine, a key marker of kidney function, was elevated at 5.7 mg/dL, confirming CKD as the definitive diagnosis [2]. 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 biochemical parameters presented in (**Table 1**). Proteinuria is a well-established independent risk factor for the progression of CKD in humans [15] and has similarly been linked to CKD progression in dogs and cats [16]. 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 [2]. Ultrasound findings revealed increased cortical thickness in the kidneys which is 7.5 mm **(Figure 2)**, which was suggestive of chronic kidney disease.

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Table 1: Biochemical Parameters of Blood and Urine in Dog affected from Chronic Kidney Disease and Dilated Cardiomyopathy

Name of test	Result	Reference value
Serum analysis		
Calcium(mg/dL)	9.3	9.4-11.1
Phosphorus(mg/dL)	13.2	2.7-5.4
Potassium(mEq/L)	4.7	4.1-5.4
Glucose(mg/dL)	110.3	68-104
Total protein(g/dL)	11.8	5.5-7.2
Albumin(g/dL)	2.5	3.2-4.1
Globulin(g/dL)	9.3	1.9-3.7
SGPT(U/L)	65	17-95
SGOT(U/L)	80	18-56
ALP(U/L)	12	7-115
BUN (mg/dL)	75	9-26
Ceatinine(mg/dL)	5.7	0.6-1.4
Cardiac Troponin I (ng/ml)	0.39	0.05-0.24
Urine analysis		
pH	6.5	6.0-7.0
Specific gravity	1.012	1.001-1.065
Glucose (mg/dl)	200	180-220
Proteinuria(gm/dl)	4.0	0.5-1.0

The elevated cardiac-specific troponin I (cTnI) 0.39 ng/ml in serum concentration , exceeding the normal reference value 0.05-0.24 ng/ml [17]. X-ray imaging revealed enlargement of the heart (**Figure 1)**. This indicates the presence of cardiomyopathy. In humans, cardiovascular complications are a significant cause of morbidity and mortality in CKD patients [18]. While 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 [19]. CvRD secondary to kidney disease (CvRDk) has been suggested to involve harmful interactions between the cardiovascular and renal systems, although direct evidence in

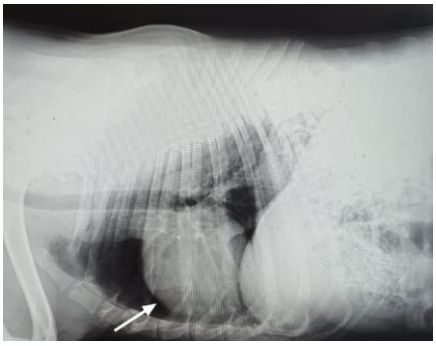

Figure 1: X-ray of Dog (Enlarged Heart)

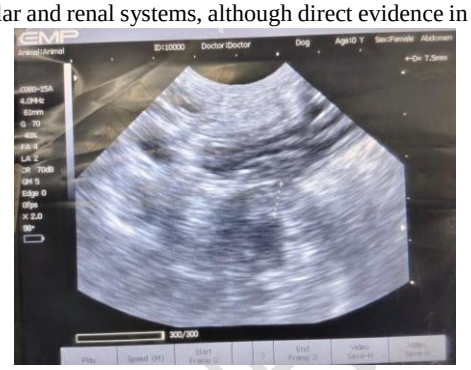

Figure 2: Ultrasound of Dog (Increased Renal Cortex Size)

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 [20]. These findings are consistent with previous studies demonstrating the connection between cardiomyopathy and CKD.

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 [21].

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

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 10.9 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

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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 [22]. 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 [23]. For hypertension management, Traditionally induced by CKD in dogs consists of (i) a salt restriction diet and

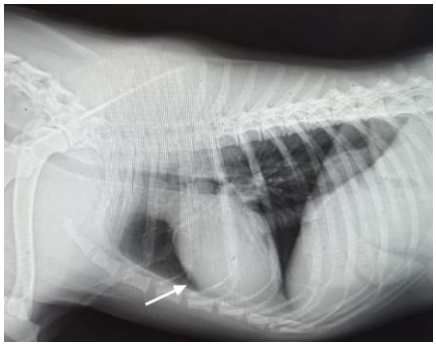

Figure 3: X-ray of Dog (Heart Size Returns to Normal)

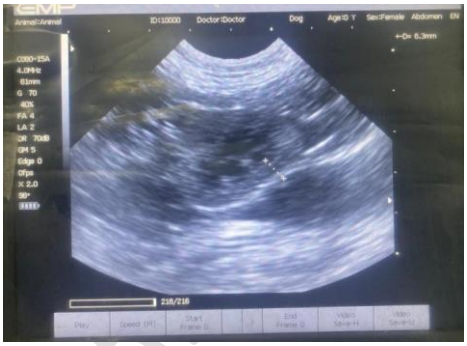

Figure 4: Ultrasound of Dog (Renal Cortex Size Returens to Normal)

diuretics to reduce extracellular fluid, (ii) adrenergic blocking agents like beta-adrenergic receptor blockers (e.g., propranolol, atenolol) to decrease heart rate and alpha-adrenergic receptor blockers (e.g., phenoxybenzamine, prazosin) to induce vasodilation, (iii) angiotensin-converting enzyme (ACE) inhibitors (e.g., benazepril, enalapril) to reduce circulating angiotensin-II, a potent vasoconstrictor, (iv) calcium channel blockers (e.g., amlodipine) to promote vasodilation [24]. Here amlodipine, a calcium channel blocker, was administered 0.2-0.4 mg/kg once daily, which proved to be effective. Amlodipine is often the first-choice drug for treating hypertension in cats, though clinical data on its efficacy in hypertensive dogs are limited. In cases where ACE inhibitors (Benazepril: 0.25 to 0.5 mg/kg orally once a day) alone are insufficient, amlodipine is commonly added [20]. For improvement of dilated cardiomyopathy 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 [2] , which included appetite stimulants, phosphate binders, and a renal diet. Studies have shown that renal diets significantly reduce the risk of uremic crisis 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 [25]. During the two-month follow-up period, no complications were reported, and the dog was found to be in good health, with all blood and urine test parameters returning to normal ranges. (Table 3). and renal cortex diameter returns to normal at 6.3mm (Figure 3) and atirium size become normal (Figure 4). The results indicate a positive response to the treatment.

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Table 3: Biochemical Analysis of Blood and Urine in Dog Recovered from Chronic Kidney Disease and Dilated Cardiomyopathy

Name of test	Result	Reference value
Serum analysis		
Calcium(mg/dL)	9.7	9.4-11.1
Phosphorus(mg/dL)	5.6	2.7-5.4
Potassium(mEq/L)	4.8	4.1-5.4
Glucose(mg/dL)	109	68-104
Total protein(g/dL)	7.8	5.5-7.2
Albumin(g/dL)	2.9	3.2-4.1
Globulin(g/dL)	4.3	1.9-3.7
SGPT(U/L)	70	17-95
SGOT(U/L)	49	18-56
ALP(U/L)	21	7-115
BUN (mg/dL)	29	9-26
Ceatinine(mg/dL)	1.5	0.6-1.4
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	180-220
Proteinuria(gm/dl)	1.3	0.5-1.0

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Conclusion

Chronic Kidney Disease (CKD) is a prevalent and progressive condition in dogs, particularly affecting older animals but with cardiomyopathy is not common. This report highlights the importance of early diagnosis and comprehensive management in improving the prognosis of CKD, especially when complicated by secondary conditions such as Dialated cardiomyopathy. 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 and cardiomyopathy.

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