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Canine Chronic Kidney Disease (CKD): Insights into Diagnosis, Management and Prognosis in Pit Bull Dog: A Case Report

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ABSTRACT

The Study was conducted from June to July, 2023 at Teaching Veterinary Clinical Complex Rampura Phul, Bathinda, Punjab, India to study the overview of CKD in dogs, including its etiology, clinical presentation, diagnostic approach, management strategies, and prognostic factors. A male Pit bull dog, aged five and a half years, weighing 14.5 kg was presented with deteriorating body condition, pale gums, lethargy, weakness, fever, and low appetite. Laboratory tests such as serum chemistry (e.g., creatinine, blood urea nitrogen), urinalysis (e.g., proteinuria, urine specific gravity), and imaging studies (e.g., ultrasound) were done. Elevated levels of BUN, Creatinine, Potassium and proteinurea were suggestive of CKD. Further, the kidney lacked cortico-medullary differentiation. Urinary bladder concretions were also observed on USG examination. These alterations confirmed cholecystitis or chronic kidney disease. The objective of study was to manage CKD in the patient which aimed to halt the spread of the illness, reduce symptoms and improve quality of life through dietary modifications (e.g., low phosphorus, high-quality protein), medications and fluid therapy to restore electrolyte balance. As a result of regular monitoring of clinical signs, renal function, treatment and follow up for 2 months azotaemia was resolved to quite extent with restoration of blood urea nitrogen and creatinine upto normal reference level and improvement of dog was also evident by normal physical activity, diet intake and playful behaviour.

KEYWORDS: CKD, creatinine, diagnosis, electrolyte, management, multidisciplinary, polyuria, ultrasound

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1. INTRODUCTION

Renal diseases in veterinary medicine are categorized as chronic kidney disease (CKD) and acute kidney injury (AKI). Chronic kidney disease (CKD) is characterised by structural or functional changes in one or both kidneys that last longer than three months (Polzin, 2011) and clinical signs usually occur when more than 70.0% of nephrons are compromised (Pedrinelli et al., 2020). This triggers glomerular capillary hypertension, and vasoconstriction of the afferent arteriole, which lowers the glomerular filtration rate, causes renal azotemia, and impairs urine concentration (Reynolds and Lefebvre, 2013; Vázquez-Manzanilla et al., 2023). With a prevalence estimated in dogs of up to 7%, it is the most common kidney disease in small animals (Lund et al., 1999). It affects dogs of all ages, but older dogs are affected more frequently. CKD can be congenital or acquired, the latter being more common in animals of more than 7 years of age (Pedrinelli et al., 2020). In daily small animal practice, ICKD is regarded as a common pathology. Once parenchymal mass loss reaches a critical level, the condition tends to worsen and become irreversible (Polzin, 2011; O'Neill et al., 2013). Numerous diseases, occurrences, and other variables can contribute to it, and various clinical results can be utilized to gauge its severity (Polzin, 2011). Changes in the equilibrium of water, electrolyte, acid-base, hormonal, haematological, and cardiovascular processes are hallmarks of the clinical condition known as renal insufficiency (Pathak et al., 2023). Renal insufficiency, which can progress to renal failure, can be caused by a number of factors, including ischaemia, nephritis, urinary tract blockage, lower urinary tract infections, and other conditions that result in decreased renal perfusion (Karunanithy et al., 2019). The presence of anaemia, low body condition score, proteinuria, hypertension, and hypoalbuminemia are recognized markers linked to the course and prognosis of CKD in canines. (Bartges, 2012; Parker and Freeman, 2011 and Jacob et al., 2005). Various factors have been linked to the pathophysiology of chronic kidney disease (CKD), such as infections (chronic pyelonephritis), nephrotoxicity, neoplasia, prior acute kidney injury (AKI), or urinary obstruction. However, the etiology of CKD is frequently unclear and stays that way throughout the course of the disease (O'Neill et al., 2013; Cowgill et al., 2016 and Rudinsky et al., 2018). Creatinine and Blood urea nitrogen are the functional marker for kidney failure i.e., specific to the kidney and safe as well as affordable to be used in routine clinical settings (Pathak et al., 2023).

Though chronic kidney disease is a progressive disorder, early detection and management helps to minimise disease progression rate and improve patient life expectancy (O'Neill et al., 2013). Due to the shortcomings of the diagnostic indicators that are currently available, CKD is frequently detected late in the disease's progression, when compensatory mechanisms are exhausted and irreversible, severe renal parenchymal damage has already taken place. Additionally, it is well recognized that a worse prognosis is linked to a delayed diagnosis of CKD. The aforementioned factors provide sufficient justification for concentrating all available resources on prompt diagnosis, which promotes the stability of the disease, slows its course, and extends patient survival (Grauer, 2005). So, the present study was planned with the objective to identify and diagnose CKD by multidisciplinary approach for optimal diagnosis, management, and monitoring and to slow down its progression through dietary modifications (low-protein, low-phosphorus diets), medications and fluid therapy to correct dehydration and electrolyte imbalances.

2. MATERIALS AND METHODS

2.1. Case description

This case reported on June, 2023 at Teaching Veterinary Clinical Complex Rampura Phul, Bathinda, Punjab, described the clinical appearance, diagnostic assessment, treatment, and prognosis of a dog diagnosed with CKD.

2.2. Patient information

A male Pit bull, aged five and a half years, weighing 14.5 kg, was presented on June, 2023 at Teaching Veterinary Clinical Complex Rampura Phul, Bathinda, Punjab with deteriorating body condition, pale gums, lethargy, weakness, fever, and low appetite.

2.3. Diagnostic evaluation

Physical examination, including assessment of vital signs, hydration status, and body condition score, was conducted upon presentation.

2.4. Diagnostic tests performed included

2.4.1. Complete blood count (CBC)

Hematology was performed by taking 4–5 ml blood sample from cephalic vein of affected dog in Ethylenediaminetetraacetic acid (EDTA) vacutainers and blood parameters viz., Hemoglobin (Hb), Packed Cell Volume (PCV), Total erythrocyte count (TEC), Mean corpuscular volume (MCV), Mean corpuscular hemoglobin concentration (MCHC), Platelet count and Total Leukocyte count (TLC) were estimated using automatic machine ADVIA 2120 Hematology System, Siemens Healthcare Diagnostics Inc. Deerfield, IL, USA and Orphee Mythic 18–VET whereas after staining blood smears with Leishman stain, the differential leukocyte count (DLC) was manually performed by pathologist (Jain, 1986). Blood samples of the dog were taken fortnight for two months.

2.4.2. Serum biochemistry

Biochemical analysis including serum creatinine, blood urea nitrogen, electrolytes viz Na+, K+, and Cl was performed in Dry chemistry analyser (M/s Fujifilm India Pvt. Ltd. a fully owned Indian subsidiary of Fujifilm Corporation, Japan, Model: NX 600 V (Veterinary)) using commercially available Dry Chemistry slides from Fuji Film following the standard company protocols.

2.4.3. Urinalysis

Including urine specific gravity, and proteinuria assessment was done by using Plastic H_{10} reagent strips for urinalysis manufactured by DIRUI Industrial Co., Ltd, China.

2.4.4. Abdominal x-ray and ultrasound examination

The abdominal X-ray and USG of dog was done using an Allengers 150 mA X-ray Machine with CR system from Konica Minolta, Japan and USG IMV-Mindray Vetus-8, manufacturer, China, respectively.

2.4.5. Parasitological examination

A sample of 1–2 ml of blood was taken from the cephalic vein of the dog. A thin blood smear was made, and the slide was checked under a microscope for hemoprotozoan infections after being stained with Leishman's stain.

2.5. Diagnostic criteria

Diagnosis of CKD was based on persistent renal dysfunction, characterized by elevated serum creatinine levels (>1.6 mg dl^{-1}), proteinuria (>0.5 g dl^{-1}), and evidence of renal damage on ultrasound (e.g., decreased renal size, irregular corticomedullary differentiation).

2.6. Management approach

The dog was managed according to standard treatment protocols for CKD. Dietary modifications viz transition to a renal-supportive diet with controlled phosphorus and high-quality protein content and administration of fluids to maintain hydration and support renal function was done. Renal function parameters and clinical signs were regularly monitored.

2.7. Follow-up and outcome

The dog was followed up at regular intervals (e.g., every 2 weeks upto 2 months) to assess response to treatment, adjust management strategies as needed, and monitor disease progression. The outcome was evaluated based on clinical improvement, stabilization of renal function, and owner satisfaction with the management plan.

3. RESULTS AND DISCUSSION

3.1. Pathological observations

On day 1, hematological examination revealed leukocytosis with absolute neutrophilia. In addition to this, Hb, PCV and TEC were decreased suggesting anemia. Value of MCV and MCHC revealed normocytic normochromic anemia and further normocytic hypochromic anemia from 15 days till 60 days. No reticulocytes and nucleated series of RBC's were observed suggesting non-regenerative anemia (Hodges and Christopher, 2011). TLC was gradually decreased at the start of treatment and returned to normal on days 15, 30, 45, and 60, respectively. However, lymphocyte count was showing increase in its counts suggesting relative lymphocytosis. Further, Hb value, PCV and TEC were decreased fortnight as represented in Table 1. Thrombocyte count was also gradually decreased suggesting thrombocytopenia on day 30, 45 and 60.

Leukocytes are cellular biomarkers in the diagnosis of inflammation and/or infections that produces an acute response, which accelerate the process of neutrophilopoiesis, stimulating the release of PMNs, resulting in the mobilization of large amounts of neutrophils (Banga et al., 2020). Neutrophilic leukocytosis can be due to stress reaction in kidney disease (Maria, 2016). Relative

Table 1: Hematological values of affected dog at different days

	Reference value	Day 1	Day 15	Day 30	Day 45	Day 60
Hb (g %)	12–18	8.8	7.5	3.1	6.3	4.0
TLC (10^3 μl^{-1})	6–17	19	12.1	10.7	8.2	7.6
TEC (X 10^6 μl^{-1})	5.5–8.5	3.4	3.52	1.77	3.55	2.22
PCV (%)	33–55	25.0	23.7	10.7	21.4	13.5
MCV (fl)	60–77	73.52	67.32	60.46	60.28	60.81
MCHC (g %)	31–34	35.2	31.64	28.97	29.43	29.62
Platelets (10^5 μl^{-1})	2–9	6.97	2.03	1.03	1.01	0.73
Neutrophils (%)	60–72	86	68	62	64	66
Lymphocytes (%)	12–30	12	30	38	34	32
Eosinophils (%)	2–10	02	02	02	02	02

lymphocytosis in the present case might be associated with acute stress response (physiologic lymphocytosis) and chronic inflammation (Maria, 2016). Further, non-regenerative normocytic normochromic anemia may probably be due to bone marrow disease, kidney disease, exposure to toxins, parvovirus, chemotherapy drugs and other certain medications (Anonymous, 2024). However, Radiograph, Ultrasound, biochemical examination and urine examination revealed kidney disease that might be the main reason of anemia due to poor erythropoietin production by affected kidney, nutritional imbalances because of anorexia or inappetence, blood loss from uremic gastroenteritis as reported by Polzin et al., 2005. As uremic toxins build up, thrombocytopenia in the later stages of CKD may be caused by inadequate thrombopoietic activity (Habib et al., 2017).

3.2. Biochemical observations

Dogs with CKD may experience a variety of biochemical alterations that reflect the underlying causes and progression of the condition. These alterations may offer insightful information into the mechanisms involved in CKD development. The primary biochemical change in CKD is the decline in renal function, which leads to impaired filtration and excretion of waste products. As kidney function deteriorates, blood urea nitrogen (BUN) and creatinine levels rise, indicating decreased clearance of these waste products from the bloodstream. CKD can disrupt the balance of electrolytes such as sodium, potassium, and calcium in the blood. Elevated potassium levels (hyperkalemia) are common in advanced CKD stages due to reduced potassium excretion by the kidneys. Conversely, sodium and calcium levels may decrease, especially in cases of excessive urinary losses. Hyperkalemia can lead to health risks, including cardiac arrhythmias. Hyperkalemia associated with oliguria and anuria may be noted in terminal Stage 4 or whenever marked prerenal azotemia is concurrent with CKD. Systemic hypertension and associated complications develop in ~20% of affected cats and dogs and can occur at any stage (Anonymous, 2024). The kidneys play a vital part in maintaining acid-base balance in the body. In CKD, the kidneys' ability to excrete hydrogen ions and reabsorb bicarbonate is impaired, leading to metabolic acidosis. This acid-base imbalance can contribute to clinical signs such as weakness, vomiting, and muscle wasting. Protein loss in the urine (proteinuria) is a common finding in CKD. The damaged kidney filters may allow proteins to leak into the urine, resulting in decreased serum protein levels and potential complications such as edema and impaired immune function. CKD-related changes in erythropoietin production by the kidneys can lead to anemia, leading to fatigue, pale mucous membranes, and exercise intolerance. Diagnostics in Stages 1 and 2 are frequently overlooked or made by accident during imaging tests or urine analyses carried out for unrelated reasons. The BUN, serum creatinine, and inorganic phosphorus concentrations rise in Stages 3 and 4. Perini-Perera et al., 2021 noticed that GFR markers viz serum Creatnine and serum urea exhibited differences between early and advanced CKD stages, associated with the different degree of renal function decline present in each CKD stage.

Renal biochemical markers, such as creatinine and BUN, were noticeably elevated on Day 1, 15 and 30 (Table 2). Potassium level also elevated likewise. (Table 2), suggesting chronic kidney disease. Although, there was no discernible change in the sodium and chloride levels throughout the study period. Similar findings were reported by Vazquez-Manzanilla et al. (2023) in dogs suffering with CKD. Additionally, significantly higher levels of BUN and creatinine were noted by Dunaevich et al., 2020. Elevated BUN levels are often a key indicator of CKD progression. Serum creatinine level is elevated when there is a significant reduction in the glomerular filtration rate or when urine elimination is obstructed. About 50% of kidney function must be lost before arise in Serum creatinine can be detected thus Serum creatinine is considered as a late marker of acute kidney injury. However, BUN, Creatinine and Potassium levels began to decrease after 30 days' post treatment.

3.2.1. Urinary examination observations

Depending on the body's requirements for maintaining water homeostasis, the specific gravity of urine in dogs and cats can range from 1.001 to 1.060 and 1.005 to 1.080, respectively. Urine specific gravity in dehydrated animals

Table 2: Serum biochemical values observed in dog at different days

	*Reference value	Day–0	Day–15	Day–30	Day–45	Day–60
BUN (mg dl^{-1})	8–28	104.5	98.1	55.4	48.1	35.2
Creatinine (mg dl^{-1})	0.5–1.7	16.9	14.61	7.22	7.13	4.2
Na (mEq l^{-1})	142–152	126	142	144	119	131
K (mEq l^{-1})	3.9–5.1	5.5	6.4	5.7	4.3	3.8
Cl (mEq l^{-1})	110–124	88	108	108	87	109

Na: sodium; K: Potassium, Cl: Chloride

with appropriate renal function should be greater than 1.035 in cats and greater than 1.030 in dogs. An early indicator of CKD is the inability to generate concentrated urine in the face of dehydration. However, dogs with primary glomerular disease and certain cats may develop azotemic conditions without losing the ability to concentrate urine to a specific gravity greater than 1.035. Nevertheless, in an animal with azotemia of renal origin, concentrated urine is rarely observed when the serum creatinine is greater than 4 mg dl^{-1} (Anonymous, 2024). The urine sample that was centrifuged revealed a significant increase in waxy casts per hpf and Lpf. Protein was also observed in urine sample indicating CKD.

3.2.2. X-ray observations

The kidneys are seen in the mid-dorsal abdomen just caudal to the stomach. The right kidney is slightly cranial to the left one. The left lateral and ventrodorsal views are best to individually visualise the kidneys. Kidneys may be abnormal in size, shape or opacity. The ureters and urethras are not visible on plain radiographs of a healthy patient, but they can be visualised using contrast radiography. The bladder is round and seen in the caudal ventral abdomen, below the colon (Anonymous, 2024). The X-ray was conducted at 150 mA, 72 KvP, and 10 mAS presented tiny radio dense concretions in the bladder area indicating urinary calculi, also termed as bladder stones or uroliths. These may be mineralized deposits that can form in the bladder due to various factors such as mineral imbalances, urinary tract infections, or metabolic disorders.

3.2.3. Ultrasonography observations

High frequency sound waves are used in ultrasound to produce a "picture" of an interior structure. The waves are emitted by a probe that the doctor holds in their hand and scans over the area of interest. These waves can be reflected back, pass through, or absorbed by tissues all of which help to create a picture. Ultrasound is considered a non-invasive test and can be done on any number of body areas. The USG (IMV-Mindray Vetus-8, manufacturer, China) revealed a dilated gall bladder with sludge in the duct. Stomach was gas-filled and watery. The kidney lacked cortico-medullary differentiation. Urinary bladder concretions were discovered. These alterations pointed to cholecystitis or chronic kidney disease. Huguet et al., 2023 observed loss of corticomedullary distinction in dogs and cats suffering with CKD. Similarly, Perondi et al., 2020 reported increased cortical echogenicity and abnormal ratio of cortico-medullary junction in dogs with CKD.

3.2.4. Parasitological observations

Canine babesiosis commonly results in acute renal failure as one of its most common consequences. This complication leads to reduction in the glomerular filtration rate and in consequence causes azotemia and uremia (Winiarczyk et al., 2017). However, the dog was found negative for the same.

3.2.5. Treatment and recovery

Therapeutic management was commenced with initial fluid therapy with Ringer's Lactate @ 200 ml IV followed by DNS @ 200 ml IV, o.d. for three days along with oral diuretic (Tab.®Lasix 40 mg @ 1 tab b.i.d.) and antacid (Sodabicarb @ 1 tsf b.i.d.) to combat metabolic acidosis. Three days, post therapy, dose of DNS was augmented to 500 ml IV along with other medications at the same pace for the next 15 days. Dog exhibited remarkable improvement during period of treatment and after 15 days of therapy 500 cc of Ringer's Lactate was also included in therapy for next 15 days along with subcutaneous administration of erythropoietin @ 100 IU kg^{-1} at weekly interval upto four shots for management of anaemia. The animal responded well to a 15 day repetition of this treatment with clinical moderate restoration of appetite and activity. Eventually, after 45 days, a similar course of treatment was continued for the following 15 days, with a gradual halving of the dosage. Following this, the animal began consuming food and water along with weight gain and the fluid therapy and other medications were discontinued at this stage. Additionally, oral medication with Renal Essentials (Veteriscience laboratories, USA) @ 1 tab. Was given each day for a week to regain optimum renal function and improving mineral imbalance. As a result, azotaemia was resolve with restoration of blood urea nitrogen and creatinine upto normal reference level and recovery of dog as evident by normal physical activity and playful behaviour.

4. CONCLUSION

CKD in this case was diagnosed through clinical signs, laboratory evaluations and imaging studies, which emphasized the value of a multidisciplinary approach for optimal diagnosis, management, and monitoring. Though CKD was not completely cured, but its progression was slowed through dietary modifications and fluid therapy to correct dehydration and electrolyte imbalances. Thus, through comprehensive care and vigilant monitoring, even a progressive condition like CKD was managed to ensure improved quality of life and meaningful extensions of survival in the dog.

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