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Comprehensive clinical, hematobiochemical, urinary, and ultrasonographic profiling of canine chronic kidney disease: A multimodal diagnostic evaluation

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Abstract
Chronic kidney disease (CKD) is a progressive and irreversible condition frequently encountered in canine practice. This study aimed to determine the prevalence of CKD in dogs and to evaluate the associated clinical signs and hematological, biochemical, urinary, and ultrasonographic alterations. A total of 3, 180 dogs presented to the Veterinary Clinical Complex, Khanapara, were screened over a six-month period, and 40 were confirmed with CKD, yielding an overall prevalence of 1.25%. Labrador Retrievers, male dogs, and animals aged >6-10 years showed the highest prevalence. The most common clinical signs included inappetence, vomiting, polyuria, polydipsia, diarrhoea, oral lesions, weight loss, and lethargy. Hematological evaluation revealed a significant increase in WBC count, while reductions in RBC, Hb, and Hct values were non-significant. Biochemical analysis demonstrated markedly elevated serum creatinine, BUN, phosphorus, and potassium, accompanied by decreased albumin and chloride levels. Urinalysis showed amber to deep amber urine, presence of crystals, RBCs, WBCs, epithelial cells, and an increased UPC ratio, indicating proteinuria. Ultrasonographic findings included heightened renal echogenicity, loss of corticomedullary distinction, and changes in renal size, all suggestive of chronic renal pathology. Overall, the study highlights the importance of a multimodal diagnostic approach combining clinical assessment with hematological, biochemical, urinary, and imaging evaluations for the early detection and accurate staging of CKD in dogs.

Keywords: Chronic kidney disease, dogs, serum biochemistry, urinalysis, ultrasonography, prevalence

Introduction
Chronic kidney disease (CKD) is a multifactorial clinical syndrome that represents the end stage of a gradual pathological deterioration affecting one or more components of the nephron, including the glomeruli, tubules, interstitium, and vasculature (Brown, 2007) [2]. According to Polzin *et al.* (2011) [22], CKD is defined as the presence of structural or functional abnormalities in one or both kidneys persisting for at least three months. The likelihood of developing this progressive disorder increases with advancing age (Perez *et al.*, 2018) [18]. CKD is characterised by varying degrees of architectural distortion within the renal parenchyma, which may occur with or without overt clinical signs of functional impairment (Constantinescu *et al.*, 2015) [3]. Persistent proteinuria is recognised as one of the most significant risk factors contributing to the increased morbidity and mortality associated with CKD (Jacob *et al.*, 2005) [9]. The predominant clinical manifestations of uraemia include gastrointestinal disturbances such as reduced appetite, vomiting, halitosis, oral ulcerations, uraemic stomatitis, tongue tip necrosis, and melena or haematochezia. Additional common signs comprise depression, lethargy, weight loss, cachexia, hypertension, and anaemia (Kumar *et al.*, 2020; Tufani *et al.*, 2017) [14, 33]. Urine output may vary depending on the underlying cause and stage of the disease (Sheeran, 2021; Mishra, 2019) [27, 17]. Ultrasonography serves as an essential diagnostic modality for identifying structural abnormalities of the kidneys. Combined evaluation of blood and urine parameters plays a critical role in the early detection of renal dysfunction. Importantly, normal serum creatinine values do not necessarily indicate normal renal function. More sensitive indicators of glomerular filtration rate (GFR) including plasma creatinine clearance, asymmetric

dimethylarginine (ADMA), symmetric dimethylarginine (SDMA), cystatin C, neutrophil gelatinase-associated lipocalin (NGAL), kidney injury molecule-1 (KIM-1), urinary microalbumin, urinary protein-to-creatinine ratio (UPCR), and urine albumin-to-creatinine ratio (UACR) offer greater diagnostic utility, although limitations may restrict their routine clinical use (Singh, 2017) [28]. The present study outlines the prevalence of CKD in dogs and documents the associated haematological and biochemical alterations observed in cases presented to the Veterinary Clinical Complex, Khanapara, during a six-month study period.

Material and Methods

Criteria for Inclusion in Animal Selection

The study was conducted over a six-month period at the Veterinary Clinical Complex, College of Veterinary Science, Khanapara, Assam, and included dogs presented from various regions of Assam and neighboring northeastern states. Dogs of any age, breed, or sex showing clinical signs indicative of chronic kidney disease (CKD) including inappetence, vomiting, altered urination, polydipsia, anemia, lethargy, diarrhea, and oral lesions were screened through physical examination, hematological analysis, serum biochemical profiling, ultrasonography, and urinalysis to confirm CKD (Brown *et al.*, 2007) [2].

Sample collection

On day 0 (pre-treatment), 2 mL of blood was collected into a K_2-EDTA vial and 3 mL into a clot-activator Vacutainer for hematological and biochemical analyses. Additionally, 20-25 mL of urine was aseptically collected by catheterization using an appropriately sized infant feeding tube for routine examination.

Evaluation of Hematological and Serum Biochemical Parameters

Hematological analyses were performed using an automated hematology analyzer (MS4Se, MELET SCHLOESING Laboratories) to determine WBC count (m/mm³), RBC count (M/mm³), hematocrit (Hct, %), and hemoglobin (Hb, g/dL). Serum biochemical parameters were assessed using a clinical auto-analyzer (Idexx Catalyst One), which measured creatinine, blood urea nitrogen (BUN), total protein, albumin, phosphorus, calcium, potassium, sodium, and chloride concentrations.

Urine analysis

Urine samples were evaluated using Nucleus Diagnostics Nuvision test strips with the Nuvision urine strip analyzer. Color and clarity were assessed visually, followed by analysis of pH, urine specific gravity (USG), urine protein, urine creatinine, and microscopic sediment examination. The urine protein-to-creatinine (UPC) ratio was calculated using the formula:

$$\text{Urine protein and creatinine (UPC) ratio} = \frac{\text{Urinary Total protein (mg/dl)}}{\text{Urinary creatinine (mg/dl)}}$$

Ultrasonography

A color Doppler ultrasound system (ACUSON NX3 Elite, Siemens Healthineers) was used to evaluate renal architectural changes in dogs with CKD. Both a 5-8 MHz convex probe and a 3.5-10 MHz linear probe were employed for abdominal imaging.

Data analysis: Statistical analysis of the experimental data was carried out using two-way ANOVA (SAS Enterprise Guide 9.3 version).

Results

In the present study, out of 3, 180 registered dogs, 40 were confirmed to have CKD based on clinical history, physical examination, hematobiochemical findings, urinalysis, and ultrasonographic evaluation, yielding an overall prevalence of 1.25%. The distribution of CKD cases by breed, age, and sex is shown in Figures 1-3 and tables 1-4. Labrador Retrievers, male dogs, and those aged 6-10 years were the most frequently affected groups.

Clinical Signs

The most frequently observed clinical signs were inappetence, oral lesions, vomiting, polyuria, and polydipsia. The full distribution of clinical manifestations recorded in the study is presented in Table 5 and Figure 4.

Haematology

Different hematological parameters WBC, RBC, Hct, and hemoglobin of healthy control and CKD-affected dogs are presented in Table 6. In the present study, a highly significant ($P<0.01$) increase in the Mean±SE of WBC count was observed in CKD-affected dogs (24.22±3.28) compared with healthy controls (12.06±0.67). Although the Mean±SE values of RBC, Hb, and Hct in CKD-affected dogs (RBC: 12.06±0.67; Hb: 11.23±0.47; Hct: 36.73±1.58) showed a decreasing trend relative to healthy controls (RBC: 6.67±0.27; Hb: 12.14±0.49; Hct: 41.57±1.63), these differences were not statistically significant ($p>0.05$).

Serum Biochemistry

The biochemical parameters serum creatinine, BUN, total protein, albumin, phosphorus, calcium, potassium, sodium, and chloride of healthy control and CKD-affected dogs are presented in Table 7. A highly significant ($P<0.01$) increase in the Mean±SE values of serum creatinine (5.13±0.60) and BUN (60.68±5.40) was observed in CKD-affected dogs compared with healthy controls (creatinine: 1.14±0.04; BUN: 16.10±0.95). Serum phosphorus (7.84±0.25) and potassium (5.33±0.22) levels were also significantly elevated ($P<0.05$) relative to controls (4.68±0.20 and 4.37±0.21, respectively). A significant ($P<0.05$) decrease in serum albumin (1.46±0.06) and chloride (109.37±1.17) was recorded in CKD-affected dogs compared with healthy values (2.55±0.036 and 114.00±0.92, respectively). In contrast, no significant (P>0.05) differences were observed in total protein (7.14±0.12), calcium (9.16±0.16), and sodium (148.12±0.89) when compared with the corresponding control values (7.84±0.25, 9.53±0.21, and 149.75±0.92, respectively).

Urine analysis

Urine parameters including color, pH, specific gravity, urine protein, urine creatinine, and UPC ratio are presented in Table 8. In CKD-affected dogs, urine ranged from amber to deep amber and was mostly turbid or translucent, whereas urine from healthy controls appeared colorless to pale yellow and clear. Urine pH (6.85±0.10) and specific gravity (1.010±0.004) in CKD dogs showed no significant differences compared with healthy controls (6.83±0.26 and 1.006±0.002, respectively). Microscopic examination of

CKD urine samples revealed crystals (triple phosphate, calcium oxalate, uric acid), RBCs, WBCs, epithelial cells, and occasional casts (figs 5, 6 & 7).

Nephrosonographic alterations in kidneys of CKD dogs

A nephrosonogram was performed on CKD-affected dogs to evaluate alterations in renal architecture and echogenicity. The sonographic findings are summarized in Table 9 and illustrated in Figures 8 & 9.

Discussion

In the present study, the prevalence of chronic kidney disease (CKD) among dogs was 1.25%, which was lower than the rates reported by Tufani *et al.* (2015) [32] and Karunanithy *et al.* (2019) [12], who recorded 2.58% and 2.22%, respectively. These variations may reflect differences in population size, study duration, case load, diagnostic criteria, and screening protocols across studies. Among the affected breeds, Labrador Retrievers showed the highest prevalence (0.64%), consistent with earlier findings (Tufani *et al.*, 2015; Joshi *et al.*, 2021) [32, 10]. Their predisposition may be attributed to a higher risk of systemic diseases such as cardiovascular disorders and diabetes, which serve as important comorbid risk factors for CKD. Male dogs (0.84%) were more commonly affected than females (0.41%), which aligns with previous reports (Athaley, 2018; Meena *et al.*, 2022) [1, 16]. This sex-related difference may be due to an increased predisposition of males to urolithiasis and prostate-related disorders, which can impede urine flow (Katoch *et al.*, 2017; Kumar *et al.*, 2020) [13, 14]. Behavioral factors such as heightened aggression and increased catecholamine release may further predispose males to cardiovascular stress, an additional risk factor for CKD.

Age-wise distribution showed the highest occurrence in dogs aged >6-10 years (0.72%), in agreement with earlier studies (Devipriya *et al.*, 2018; Reddy *et al.*, 2021) [4, 24]. Advancing age is strongly associated with decreased nephron number, reduced renal blood flow, and impaired tubular function, all of which contribute to CKD progression (Polzin, 1990; Grauer & Lane, 1995) [21, 6]. The clinical signs observed such as inappetence, vomiting, polyuria, polydipsia, diarrhoea, oral lesions, weight loss, and lethargy were characteristic of renal insufficiency and consistent with previously documented patterns (Kumar *et al.*, 2020; Sheeran, 2021) [14, 27]. Inappetence and vomiting may be attributable to the accumulation of uremic toxins and altered secretion of appetite-regulating hormones like leptin and ghrelin. Vomiting may also result from stimulation of the chemoreceptor trigger zone, decreased gastrin clearance, and increased gastric acidity (Tufani *et al.*, 2017; Kumar *et al.*, 2020) [33, 14]. Diarrhoea could be linked to uremic enterocolitis associated with advanced uremia (Polzin, 2011) [22]. Weight loss and lethargy likely stem from inadequate caloric intake, malabsorption, and the catabolic effects of uraemia. Pale mucous membranes likely reflect reduced erythropoietin secretion, while mucosal congestion may be due to dehydration. Polyuria and polydipsia arise from compromised urine-concentrating ability (Polzin, 2010) [23], and oral ulceration and halitosis may be caused by urease-positive oral bacteria converting urea into ammonia or direct mucosal irritation from uremic toxins (Grauer, 2009; Kumar *et al.*, 2020) [6, 14].

Hematological analysis revealed a significantly elevated WBC count in CKD-affected dogs, which may indicate systemic inflammation, urinary tract infections, or immune activation due to circulating uremic toxins (Sonu *et al.* 2018; Gupta *et al.* 2022) [29, 7]. Although RBC, Hb, and Hct values showed a decreasing trend, the changes were not statistically significant. These reductions are expected in CKD due to impaired erythropoietin secretion, shortened RBC lifespan, and nutritional deficiencies, but the non-significant differences may reflect early disease stages or compensatory mechanisms.

Serum biochemical findings showed a highly significant increase in serum creatinine and BUN, confirming reduced glomerular filtration and azotemia, characteristic of CKD (Sumit *et al.*, 2018) [30]. Serum phosphorus and potassium were significantly elevated, owing to reduced renal excretion. Hyperphosphatemia contributes to secondary hyperparathyroidism and accelerates nephron damage, while hyperkalemia poses cardiovascular risks due to its effects on cardiac electrical activity (Ribeiro *et al.* 2020 and Thade *et al.* 2021) [25, 31]. A significant decrease in serum albumin may result from protein-losing nephropathy, chronic inflammation, or malnutrition or due to increased filtration of albumin through glomeruli, owing to its molecular size (Shaw and Ihle, 2013) [26], while reduced chloride likely reflects electrolyte imbalance or metabolic acidosis due to dehydration, vomiting, polyuria, diarrhea and inadequate water consumption occurred in the majority of the CKD affected dogs (Thade *et al.*, 2021) [31]. Total protein, calcium, and sodium levels did not differ significantly, which is consistent with the compensatory mechanisms that often maintain these parameters within normal ranges during early or moderate CKD.

Urinalysis showed that CKD-affected dogs commonly had amber to deep amber, turbid urine, which may indicate dehydration, cellular debris, or poor filtering ability (Kaneko *et al.*, 2008) [11]. Urine pH and specific gravity did not differ significantly between CKD and control dogs, possibly due to variability in hydration status and disease stage (Yadav *et al.* 2020) [34]. Microscopic examination revealed the presence of crystals (triple phosphate, calcium oxalate, uric acid), RBCs, WBCs, epithelial cells, and occasional casts. These findings suggest renal tubular damage, inflammation, or secondary urinary tract infections. The increased urine protein-to-creatinine (UPC) ratio confirmed proteinuria, a key indicator of glomerular dysfunction and a predictor of CKD progression (Singh, 2017; Polzin, 2011) [28, 22].

Ultrasonographic examination demonstrated changes such as altered renal size, increased cortical echogenicity, and loss of corticomedullary distinction, which are commonly associated with chronic renal damage. These findings correlate with nephron loss, interstitial fibrosis, and progressive architectural distortion of the kidneys, as described by Grauer, 2009; Kumar *et al.* 2011 and Perondi *et al.* 2020 [6, 14, 19].

Conclusion

The study concludes that chronic kidney disease remains a significant health concern in dogs, with a prevalence of 1.25%. Breed susceptibility, male sex, and advancing age were key contributing factors. The combined evaluation of clinical signs, hematological changes, biochemical alterations, urine abnormalities, and ultrasonographic

findings offers a reliable approach for diagnosing CKD. Early identification through routine screening particularly in high-risk breeds and older dogs supports timely intervention, slows disease progression, and enhances the quality of life in affected animals. Continued emphasis on awareness, preventive care, and regular renal monitoring is crucial for effective CKD management in veterinary practice.

Acknowledgement
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Conflict of Interest
The authors declare that they have no conflict of interest regarding the publication of this manuscript.

Table 1: Prevalence of CKD in Dogs

Total no. of dogs registered	No. of affected dogs	Prevalence (%)
3180	40	1.25

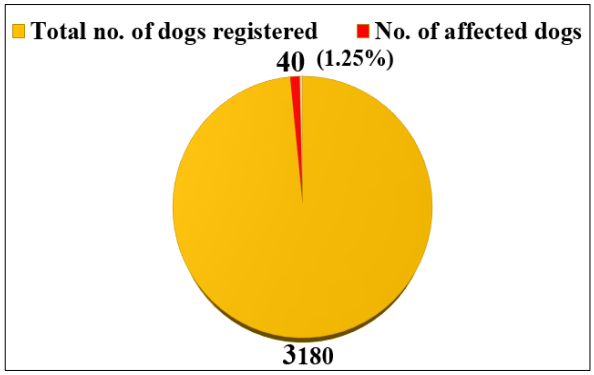

Fig 1: Pie Diagram Showing Prevalence of CKD in Dogs

Table 2: Breed-Wise Prevalence of CKD in Dogs

Breed	Total no. of dogs registered	No. of affected dogs	Prevalence (%)
Labrador Retriever	745	20	0.64
Crossbred	570	4	0.12
German Shepherd	562	4	0.12
Mongrel	652	3	0.09
Golden Retriever	254	5	0.16
German Spitz	310	2	0.06
Pit Bull	62	1	0.03
Great Dane	25	1	0.03
Total	3180	40	1.25

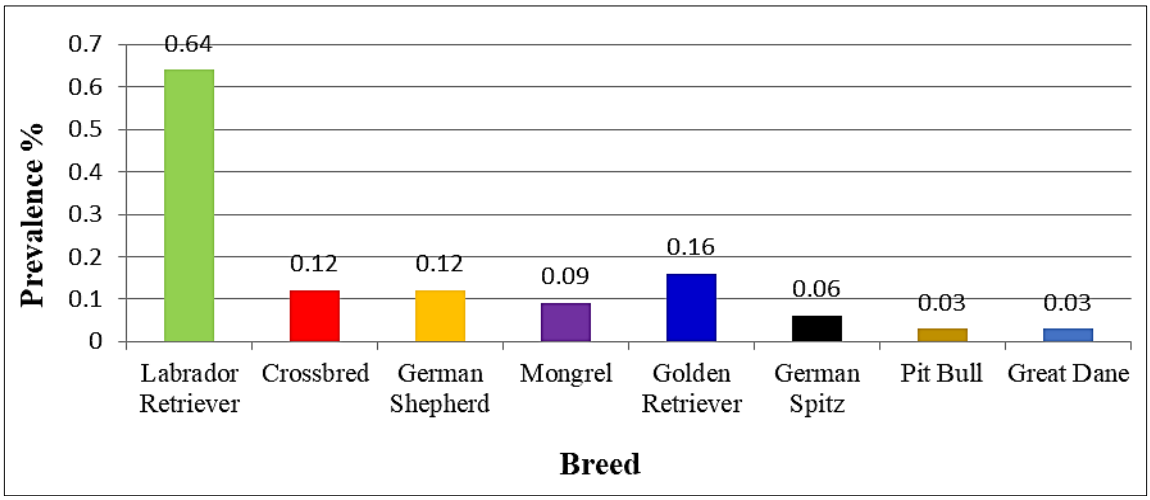

Fig 2: Bar Diagram Showing the Breed-Wise Prevalence of CKD in Dogs

Table 3: Sex-Wise Prevalence of CKD in Dogs

Sex	Total no. of dogs registered	No. of affected dogs	Prevalence (%)
Male	1792	27	1.84
Female	1388	13	0.41
Total	3180	40	1.25

TABLE 4: Age-Wise Prevalence of CKD in Dogs

Age (years)	Total No. of dogs registered	No. of affected dogs	Prevalence (%)
≤1	1140	0	0
>1-3	920	3	0.09
>3-6	495	10	0.31
>6-10	415	23	0.72
>10	210	4	0.13
Total	3180	40	1.25

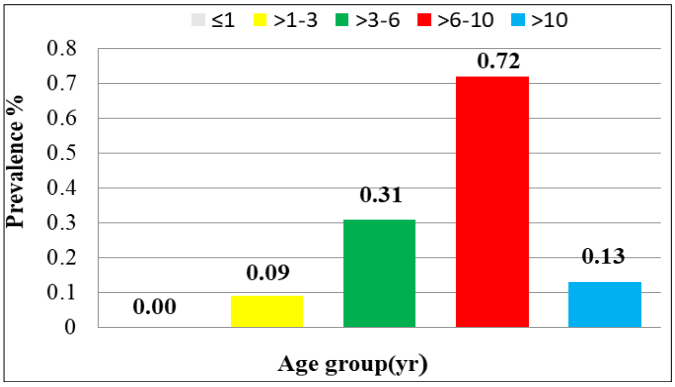

Fig 3: Bar diagram showing the age-wise prevalence of CKD in dogs

Table 5: Clinical signs exhibited by CKD dogs

Clinical signs	No. of affected dogs	Percentage (%)
Inappetance	27	67.50
Oral manifestation	21	52.50
Vomition	20	50.00
Polyuria	19	47.50
Polydipsia	17	42.50
Diarrhoea	14	35.00
Pale mucosal membrane	11	27.50
Congested mucosal membrane	9	22.50
Dehydration	11	27.50
Lethargy	11	27.50
Weight loss	6	15.00
Fever (>102.5˚)	5	12.50
Hematuria	1	2.50

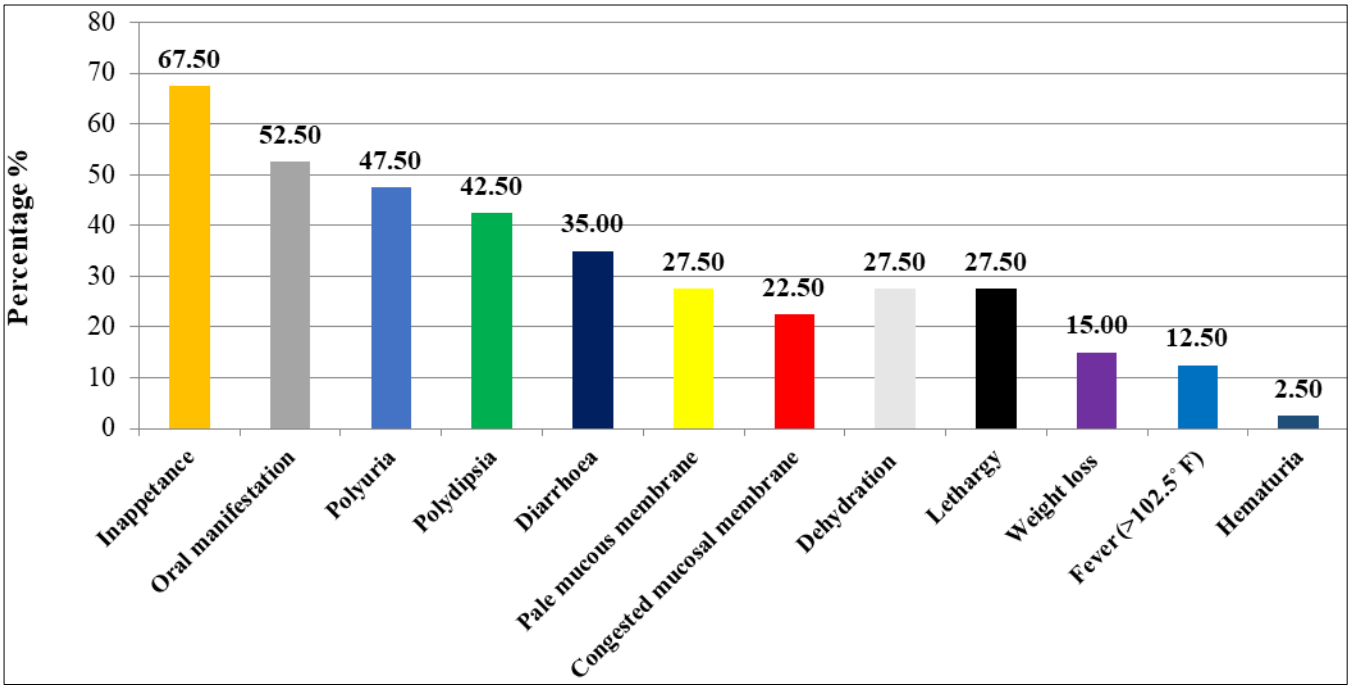

Fig 4: Bar diagram showing clinical signs exhibited by CKD dogs

Table 6: Haematological Parameters in Healthy and CKD Dogs

Parameters	Healthy CONTROL (n=20)	CKD Affected (n=40)
White blood Cell (10^3/µl)	12.06±0.67 (6.0 to 17.0)	24.22±3.28**
Red blood cell (10^6/µl)	6.67±0.27 (5.5 to 8.5)	6.066±0.20NS
Hematocrit (%)	41.57±1.63 (35.0 to 55.0)	36.73±1.58NS
Haemoglobin (g/dL)	12.14±0.49 (10.0 to 18.0)	11.23±0.47NS

**-- statistically highly significant (P ≤ 0.01) *-- statistically significant (P ≤ 0.05)
NS– non significant

Table 7: Serum Biochemical Parameters in Healthy and CKD Dogs

Parameters	Healthy Control (20)	CKD Affected (40)
Creatinine (mg/dL)	1.14±0.04 (0.5 to 1.8)	5.13±0.60**
BUN (mg/dL)	16.10±0.95 (7 to 27)	60.68±5.40**
Total Protein (g/dL)	7.84±0.25 (5.2 to 8.2)	7.14±0.12NS
Albumin (g/dL)	2.55±0.036 (2.3 to 4.0)	1.46±0.06*
Phosphorus (mg/dL)	4.68±0.20 (2.5 to 6.8)	7.84±0.25*
Calcium (mg/dL)	9.53±0.21 (7.9 to 12.0)	9.16±0.16NS
Potassium (mmol/L)	4.37±0.21 (3.5 to 5.8)	5.33±0.22*
Sodium (mmol/L)	149.75±0.92 (144 to 160)	148.12±0.89NS
Chloride (mmol/L)	114.00±0.92 (109 to 122)	109.37±1.17*

**-- statistically highly significant (P ≤ 0.01) *-- statistically significant (P ≤ 0.05)
NS– non significant

Table 8: Urine Analysis for Healthy and CKD Dogs

PARAMETERS	HEALTHY CONTROL (n=20)	CKD AFFECTED (n=40)
Color	Colorless to pale yellow	Amber color to deep amber
pH	6.83±0.26	6.85±0.10NS
Specific gravity	1.006±0.002	1.01±0.004NS
Protein in urine (mg/dL)	4.50±1.18	66.67±5.55**
Urine creatinine (mg/dl)	197.70±3.95	44.71±3.44**
Urine protein and creatinine ratio (UPC)	0.023±0.31	1.49±1.61**

**-- statistically highly significant (*P*<0.01) *-- statistically significant (*P*<0.05)
NS– non significant

Fig 5: Turbid and Amber Color Urine in Dog with CKD

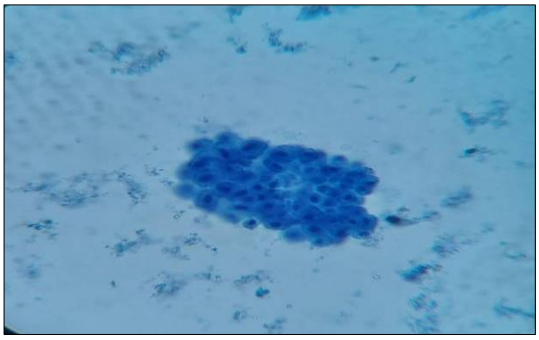

Fig 6: Typicical transitional epithelial cell stained with methylene blue (40x)

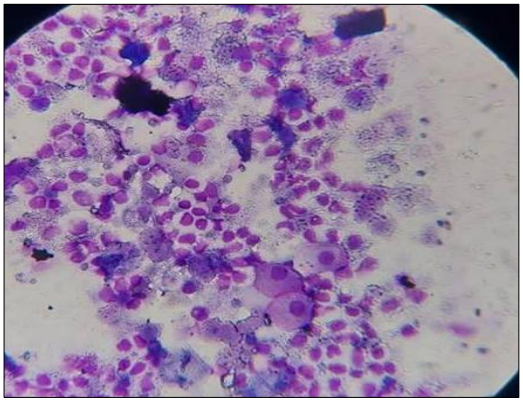

Fig 7: Epithelial cell stained with papanicolaou stain (40x)

Table 9: Nephrosonographic alteration in CKD dogs

Parameters		No. of affected dogs	Percentage (%)
Hyperechoic and thickened renal cortex		40	100.00
Cortico-medullary differentiation (CMD)	Partial loss	23	57.50
	Complete loss	17	42.50
Wavy capsule		15	37.50

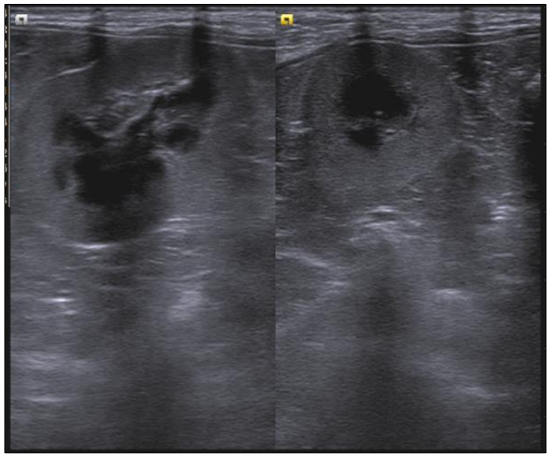

Fig 8: Longitudinal (I) and cross sectional (II) view of kidney showing increased echogenity and thickened cortex

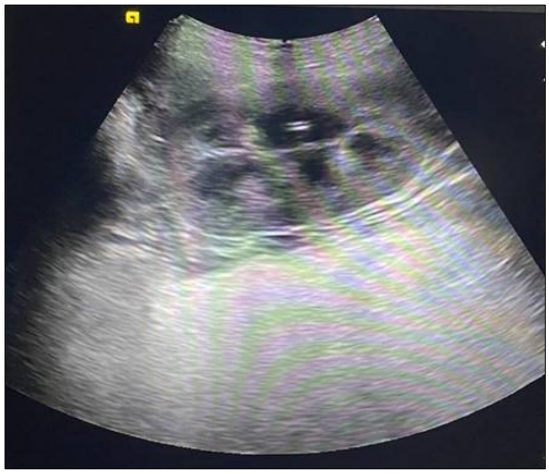

Fig 9: Kidney showing hyperechoic cortex with hazyness of the CMD and wavy capsule

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