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Study on hemato-biochemical alterations in dogs affected with renal dysfunction

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Abstract

The present study aimed to evaluate the hemato-biochemical alterations associated with renal dysfunction in dogs through comprehensive haemato-biochemical profile. A total of 57 dogs were divided into three groups: control (N=12), acute kidney injury (AKI) suspected (N=7), and chronic kidney disease (CKD) affected (N=38). Haematological evaluation revealed a significant ($p<0.01$) decrease in haemoglobin, packed cell volume, and total erythrocyte count in both AKI and CKD groups, indicating anaemia of renal origin. AKI dogs exhibited a significantly higher neutrophil percentage ($p<0.05$) suggestive of systemic inflammation. Biochemical analysis showed markedly elevated ($p<0.01$) blood urea nitrogen and serum creatinine levels in renal-compromised groups, confirming azotemia. In conclusion, renal dysfunction in dogs was characterized by anaemia and azotemia emphasizing the diagnostic importance of detailed hematobiochemical evaluation for early identification and differentiation of AKI and CKD.

Keywords: Azotemia, acute kidney injury, chronic kidney disease, hyperphosphatemia, anaemia

Introduction

Acute Kidney Injury (AKI) and Chronic Kidney Disease (CKD) are common disorders in canine medicine, and renal diseases constitute a major cause of morbidity and mortality (Krawiec, 1989; Polzin, 1995) ^[11, 19]. The progressive and irreversible loss of renal function that results in structural and functional abnormalities of one or both kidneys that last longer than three months is the hallmark of chronic kidney disease, a common clinical issue in older dogs (Polzin *et al.*, 2011; Hall *et al.*, 2018) ^[20, 10]. Nephrotoxicity, infectious agents like leptospirosis and pyelonephritis, ischemic and inflammatory injuries, metabolic disorders, and hereditary nephropathies are some of the many factors that contribute to renal dysfunction in dogs (Wettimuny, 1967; Areshkumar *et al.*, 2019; Rimer *et al.*, 2022) ^[29, 2, 23]. Reduced glomerular filtration rate and poor urine concentrating ability are two signs of age-related decline in renal function that also contribute to the incidence of disease in older dogs. According to Pradhan and Roy (2012) ^[21] and Devipriya *et al.*, (2018) ^[7], infected animals typically exhibit non-specific symptoms like anorexia, vomiting, lethargy, and pallor, along with biochemical markers of azotemia and proteinuria. Therefore, early diagnosis, differentiation, and efficient management of AKI and CKD in dogs require a comprehensive understanding of the haemato-biochemical changes linked to renal impairment.

Materials and Methods

The study was conducted on dogs referred to the Small Animal Outpatient Nephrology Unit at the Teaching Veterinary Clinical Complex, Madras Veterinary College, Chennai.

The study population consisted of both clinical cases with suspected renal impairment, based on presenting clinical signs, and apparently healthy dogs. To establish baseline reference parameters, a minimum of twelve healthy dogs of various ages, sexes, and breeds, presented for routine health check-ups and vaccinations, were randomly selected to form the control group. Clinical cases presenting to the Small Animal Nephrology Unit at the Teaching Veterinary Clinical Complex, Madras Veterinary College, Chennai, were included in the experimental group for the investigation.

Dogs in the experimental group, exhibiting clinical signs suggestive of renal failure, underwent a comprehensive evaluation including hematology, serum biochemistry and detailed clinical examination. Based on these evaluations, a minimum of forty-five dogs with a confirmed diagnosis of renal disease were selected for inclusion in the study. For both control and experimental groups, blood and urine samples were collected following standard veterinary procedures. Group I consists of 12 control group of dogs, Group II consists of 7 AKI affected dogs and 38 CKD affected dogs.

Examination of animals selected for the study

Pet owners were requested to provide detailed information regarding their dogs, including vaccination and deworming history, presence of tick infestation, changes in food intake, urine output, defecation patterns, duration of illness, prior treatments, and the occurrence of clinical signs such as vomiting, halitosis, polyuria, polydipsia, and melena. Each dog then underwent a comprehensive clinical examination, which included measurement of body temperature, assessment of skin turgor, abdominal palpation, oral cavity inspection, and evaluation of the mucous membranes for any abnormalities.

Sample Collection

About two millilitres of whole blood was aseptically collected from the saphenous vein of each dog in EDTA (Ethylene Diamine Tetra acetic Acid) vacutainers for haematological analysis. An additional two millilitres of blood was collected in clot-activator tubes for the estimation of serum biochemical parameters. Serum was separated by centrifugation at 1500 rpm for 20 minutes. To determine differential leukocyte counts, blood smears were prepared and examined according to standard protocols.

Haematological parameters such as red blood cell count (RBC count), haemoglobin (Hb), packed cell volume (PCV) and white blood cell count (WBC count) were determined using an auto haematology analyser (Mindray-BC-2800 Vet). Differential Leucocyte Count (DLC) was determined from peripheral blood smears stained with Leishman-Giemsa stain. An auto haematology analyzer used to examine haematological parameters. An automated biochemical analyzer was used to evaluate the serum biochemical parameters.

Clinical manifestations of renal disease in dogs

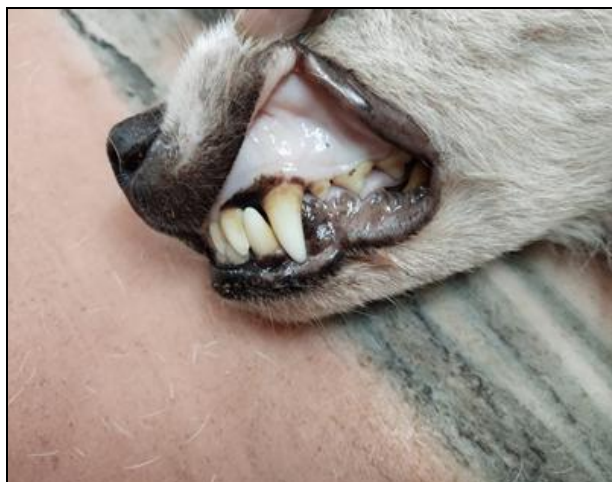


Fig 1: Pale buccal mucus membrane



Fig 2: Emaciated dog



Fig 3: Pale conjunctival mucus membrane



Fig 4: Poor skin and hair coat

Statistical Analysis

Results obtained will be statistically analysed and critically interpreted using ANOVA.

Table 1: Hemato-biochemical profile of control and renal compromised dogs

Parameters	Control Group, Group 1 (N=12), (Mean $\pm$ SE)	Renal Compromised Groups		ANOVA F-Values	P-Value
		AKI affected, Group 2 (N=7), (Mean $\pm$ SE)	CKD affected, Group 3 (N=38), (Mean $\pm$ SE)		
Hb (g/dL)	15.20 $\pm$ 0.60 ^b	9.74 $\pm$ 1.43 ^a	9.28 $\pm$ 0.58 ^a	14.32 ^{**}	0.000
PCV (%)	45.25 $\pm$ 1.67 ^b	28.95 $\pm$ 4.27 ^a	27.54 $\pm$ 1.79 ^a	13.83 ^{**}	0.000
TEC ($\times 10^6$ /mm ³)	7.21 $\pm$ 0.28 ^b	4.99 $\pm$ 0.77 ^a	4.59 $\pm$ 0.28 ^a	11.28 ^{**}	0.000
MCV (fl)	62.85 $\pm$ 0.79 ^a	60.25 $\pm$ 3.98 ^a	59.66 $\pm$ 1.08 ^a	1.05 ^{NS}	0.357
MCH (pg)	21.09 $\pm$ 0.28 ^a	20.24 $\pm$ 1.27 ^a	20.24 $\pm$ 0.37 ^a	0.67 ^{NS}	0.512
MCHC (%)	33.55 $\pm$ 0.17 ^a	33.78 $\pm$ 1.10 ^a	33.99 $\pm$ 0.33 ^a	0.23 ^{NS}	0.793
TLC (/mm ³)	11650 $\pm$ 1025.61 ^a	24285.71 $\pm$ 6755.47 ^b	17334.21 $\pm$ 1733.47 ^{ab}	3.09 ^{NS}	0.054
Neutrophil (%)	73.91 $\pm$ 1.44 ^a	80.14 $\pm$ 1.63 ^b	78.36 $\pm$ 1.03 ^{ab}	3.26 [*]	0.046
Lymphocyte (%)	20.91 $\pm$ 1.36 ^b	15.57 $\pm$ 1.65 ^a	17.04 $\pm$ 0.93 ^{ab}	2.91 ^{NS}	0.063
Monocyte (%)	4 $\pm$ 0.55 ^a	3.71 $\pm$ 0.42 ^a	4.36 $\pm$ 0.28 ^a	0.54 ^{NS}	0.586
Eosinophil (%)	1.16 $\pm$ 0.32 ^b	0.57 $\pm$ 0.20 ^a	0.23 $\pm$ 0.09 ^a	7.60 ^{**}	0.001
BUN (mg/dL)	17.9 $\pm$ 1.87 ^a	96.99 $\pm$ 21.00 ^b	67.71 $\pm$ 6.49 ^b	11.40 ^{**}	0.000
Creatinine (mg/dL)	0.93 $\pm$ 0.11 ^a	6.32 $\pm$ 1.86 ^b	5.47 $\pm$ 0.75 ^b	5.98 ^{**}	0.004
Total protein (g/dL)	7.03 $\pm$ 0.23 ^a	6.6 $\pm$ 0.42 ^a	7.07 $\pm$ 0.12 ^a	1.02 ^{NS}	0.367
Albumin (g/dL)	2.63 $\pm$ 0.16 ^b	2.144 $\pm$ 0.11 ^a	2.56 $\pm$ 0.07 ^b	2.67 ^{NS}	0.078
Globulin (g/dL)	4.39 $\pm$ 0.32 ^a	4.45 $\pm$ 0.38 ^a	4.51 $\pm$ 0.13 ^a	0.08 ^{NS}	0.922
A: G Ratio	0.67 $\pm$ 0.11 ^a	0.51 $\pm$ 0.06 ^a	0.59 $\pm$ 0.03 ^a	0.99 ^{NS}	0.377
Glucose (mg/dL)	72.5 $\pm$ 2.96 ^a	72.85 $\pm$ 5.24 ^a	75.42 $\pm$ 4.12 ^a	0.10 ^{NS}	0.903

*Significant ($p < 0.05$), **Highly significant ($p < 0.01$), NS-Non-significant, Mean having at least one common superscript does not differ at 5% level

Results

The mean $\pm$ SE values of hemoglobin concentration in control and renal compromised groups are presented in Table 1. The mean $\pm$ SE of hemoglobin concentration in control and renal compromised groups were 15.20 $\pm$ 0.60 g/dL, 9.74 $\pm$ 1.43 g/dL and 9.28 $\pm$ 0.58 g/dL, respectively. There was a high significant decrease ($p < 0.01$) in hemoglobin concentration in both the renal compromised groups when compared with control group dogs.

The mean $\pm$ SE values of PCV in control and renal compromised groups were presented in table 1. The mean $\pm$ SE values of packed cell volume in control and renal compromised groups were 45.25 $\pm$ 1.67 per cent, 28.95 $\pm$ 4.27 per cent and 27.54 $\pm$ 1.79 per cent respectively. There was a high significant decrease ($p < 0.01$) in packed cell volume in both the renal compromised groups of dogs when compared to the control group dogs.

The mean $\pm$ SE values of TEC in control and renal compromised groups were presented in table 1. The mean $\pm$ SE values of TEC in control and renal compromised groups were 7.21 $\pm$ 0.28 $\times 10^6$ /mm³, 4.99 $\pm$ 0.77 $\times 10^6$ /mm³ and 4.59 $\pm$ 0.28 $\times 10^6$ /mm³ respectively. There was a high significant decrease ($p < 0.01$) in TEC in both the renal compromised groups when compared with control group dogs.

The mean $\pm$ SE values of MCV in control and renal compromised groups were presented in table 1. The mean $\pm$ SE values of MCV in control and renal compromised groups were 62.85 $\pm$ 0.79fL, 60.25 $\pm$ 3.98fL and 59.66 $\pm$ 1.08fL respectively. There was no significant difference in MCV in both the renal compromised groups when compared with control group dogs.

The mean $\pm$ SE values of MCH in control and renal compromised groups are presented in table 1. The mean $\pm$ SE values of MCH in control and renal compromised groups were 21.09 $\pm$ 0.28 pg, 20.24 $\pm$ 1.27 pg and 20.24 $\pm$ 0.37 pg respectively. There was no significant difference in MCV in both the renal compromised groups and control groups.

The mean $\pm$ SE values of MCHC in control and renal compromised groups are presented in table 1. The mean $\pm$ SE values of MCHC in control and renal compromised groups were 33.55 $\pm$ 0.17 per cent, 33.78 $\pm$ 1.10 per cent and

33.99 $\pm$ 0.33 per cent respectively. There was no significant difference in MCHC in both the renal compromised groups and control groups.

The mean $\pm$ SE values of TLC in control and renal compromised groups are presented in table 1. The mean $\pm$ SE values of TLC in control and renal compromised groups were 11650 $\pm$ 1025.61/mm³, 24285.71 $\pm$ 6755.47/mm³ and 17334.21 $\pm$ 1733.47 /mm³ respectively. There was no significant difference in TLC in both the renal compromised groups and control group of dogs. Though mean $\pm$ SE values of TLC between renal compromised dogs and control group dogs were insignificant, proportionately higher TLC values were observed in acute kidney injury affected group.

The mean $\pm$ SE values of neutrophil per cent in control and renal compromised groups are presented in table 1. The mean $\pm$ SE values of neutrophil per cent in control and renal compromised groups were 73.91 $\pm$ 1.44 per cent, 80.14 $\pm$ 1.63 per cent and 78.36 $\pm$ 1.03 per cent respectively. There was a significant increase ($p < 0.05$) in neutrophil per cent in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of lymphocyte per cent in control and renal compromised groups are presented in table 1. The mean $\pm$ SE values of lymphocyte per cent in control and renal compromised groups were 20.91 $\pm$ 1.36, 15.57 $\pm$ 1.65 and 17.04 $\pm$ 0.93 per cent respectively. There was no significant difference in lymphocyte per cent in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of monocyte per cent in control and renal compromised groups are presented in table 1. The mean $\pm$ SE values of monocyte per cent in control and renal compromised groups were 4 $\pm$ 0.55 per cent, 3.71 $\pm$ 0.42 per cent and 4.36 $\pm$ 0.28 per cent respectively. There was no significant difference in mean monocyte per cent in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of eosinophil per cent in control and renal compromised groups are presented in Table 1. The mean $\pm$ SE values of eosinophil per cent in control and renal compromised groups were 1.16 $\pm$ 0.32 per cent, 0.57 $\pm$ 0.20 per cent and 0.23 $\pm$ 0.09 per cent respectively. There was a high significant decrease ($p < 0.01$) in eosinophil per cent in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of BUN in control and renal compromised groups are presented in table 1. The mean $\pm$ SE values of BUN in control and renal compromised groups were 17.92 ± 1.87 mg/dL, 96.99 ± 21.00 mg/dL and 67.71 ± 6.49 mg/dL respectively. There was a high significant increase ($p < 0.01$) in blood urea nitrogen concentration was observed in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of creatinine concentration in control and renal compromised groups are presented in Table 1. The mean $\pm$ SE values of creatinine concentration in control and renal compromised groups were 0.93 ± 0.11 mg/dL, 6.32 ± 1.86 mg/dL and 5.47 ± 0.75 mg/dL respectively. There was a high significant increase ($p < 0.01$) in creatinine concentration in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of total protein concentration in control and renal compromised groups are presented in Table 1. The mean $\pm$ SE values of total protein concentration in control and renal compromised groups were 7.03 ± 0.23 g/dL, 6.6 ± 0.42 g/dL and 7.07 ± 0.12 g/dL respectively. There was no significant difference in mean total protein concentration in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of albumin concentration in control and renal compromised groups are presented in Table 1. The mean $\pm$ SE values of albumin concentration in control and renal compromised groups were 2.63 ± 0.16 g/dL, 2.14 ± 0.11 g/dL and 2.56 ± 0.07 g/dL respectively. There was no significant difference in albumin concentration in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of globulin concentration in control and renal compromised groups are presented in Table 1. The mean $\pm$ SE values of globulin concentration in control and renal compromised groups were 4.39 ± 0.32 g/dL, 4.45 ± 0.38 g/dL and 4.51 ± 0.13 g/dL respectively. There was no significant difference in globulin concentration in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of albumin globulin ratio in control and renal compromised groups are presented in Table 1. The mean $\pm$ SE values of albumin globulin ratio in control and renal compromised groups were 0.67 ± 0.11 , 0.51 ± 0.06 and 0.59 ± 0.03 respectively. There was no significant difference in albumin globulin ratio in renal compromised dogs when compared to control group of dogs.

The mean $\pm$ SE values of glucose concentration in control and renal compromised groups are presented in Table 1. The mean $\pm$ SE values of glucose concentration in control and renal compromised groups were 72.5 ± 2.96 mg/dL, 72.85 ± 5.24 mg/dL and 75.42 ± 4.12 mg/dL respectively. There was no significant difference in glucose concentration in renal compromised dogs when compared to control group of dogs.

Discussion

In the present study, hematological and biochemical alterations were evaluated in dogs affected with renal disorders and compared with healthy controls to assess the impact of renal dysfunction on systemic physiology.

Hematological parameters of renal compromised group showed reduced hemoglobin (Hb), Packed Cell Volume (PCV) and Total Erythrocyte Count (TEC) and increased Total Leukocyte Count (TLC) and Neutrophils when compared to control group of dogs.

A significant reduction in hemoglobin concentration was observed in the renal-compromised groups compared with the

healthy controls (Sonu *et al.*, 2019) [27]. This reduction in hemoglobin levels may be attributed to impaired erythropoiesis and shortened red blood cell lifespan resulting from uremia. Similar findings of anemia with comparable values have been reported previously (Sharma *et al.*, 2015) [25].

Anemia is a well-recognized feature of canine kidney disease and is primarily associated with decreased erythropoietin production by the diseased kidneys, reduced RBC lifespan, and the suppressive effects of uremic toxins on erythropoiesis (Lippi *et al.*, 2021; Silverberg *et al.*, 2002) [17, 26]. Additional contributing factors include chronic blood loss, hemolysis, nutritional deficiencies, and bone marrow suppression resulting from the accumulation of uremic toxins (Dodds and Nicholls, 1983) [8]. Gastrointestinal blood loss and nutritional deficiencies have also been reported as contributing factors to anemia in chronic kidney disease (CKD), (Castaldi *et al.*, 1966) [4]. The present findings on PCV are consistent with the observations of Couto (2009) [5], who reported marked reductions in PCV values among dogs affected with chronic renal failure.

Mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC) did not differ significantly between renal-compromised and control dogs in the present study, indicating that the anemia was normocytic and normochromic in nature (Cowgill, 1992) [6]. This observation aligns with the findings of Ahn and Hyun (2013) [1], who described anemia of chronic disease in dogs with renal impairment as typically normocytic and normochromic, consistent with non-regenerative anemia due to insufficient erythropoietin stimulation. Kralova (2010) [14] similarly reported that normocytic, normochromic anemia is the most commonly observed hematological alteration in dogs affected with chronic renal failure.

Leukocytosis observed in renal-compromised dogs was consistent with the findings of Devpriya *et al.* (2018). Robinson *et al.*, (1989) [24] reported elevated total leukocyte counts in dogs suffering from chronic renal failure, which has been attributed to primary inflammatory diseases of the urinary system and the involvement of other organs and tissues (Osborne *et al.*, 1972) [18]. Neutrophil percentages were significantly elevated in renal-compromised groups, particularly in dogs with acute kidney injury (AKI), indicating a stress leukogram and systemic inflammation (Lippi *et al.*, 2021) [17]. Lymphocyte and monocyte percentages did not differ significantly between groups, whereas eosinophil counts were markedly reduced, possibly due to stress-induced corticosteroid release or the effects of uremic toxins. Mild lymphopenia observed in the AKI group compared with controls is consistent with previous observations (Kralova *et al.*, 2010) [14].

Biochemically, blood urea nitrogen (BUN) and serum creatinine concentrations were significantly elevated ($p < 0.01$) in both renal-compromised groups compared with healthy controls, reflecting impaired glomerular filtration rate (GFR). The increased urea and creatinine levels in renal failure may result from a pronounced decline in GFR, reduced renal excretion, increased tubular reabsorption of urea, and a diminished capacity of the kidneys to eliminate proteinaceous catabolites (Osborne *et al.*, 1972) [18]. Similar observations have been reported by Smith and Jones (2019), who demonstrated that progressive renal dysfunction leads to the accumulation of nitrogenous waste products in the bloodstream. Elevated BUN and creatinine values serve as

key indicators of renal insufficiency and are widely accepted diagnostic markers for CKD and AKI (Lee *et al.*, 2012; Ahn and Hyun, 2013; Pradhan and Roy, 2012; Puri *et al.*, 2015) [15, 1, 21, 22].

Total protein, albumin, globulin, and albumin-to-globulin (A:G) ratio values did not differ significantly among the groups, suggesting minimal alteration in hepatic protein synthesis or urinary protein loss in the studied stages. Mild hypoalbuminemia observed in some cases may be attributed to proteinuria or reduced protein intake (Sonu *et al.*, 2019) [27]. Mean albumin values were generally lower than the reference range, possibly due to gastrointestinal or renal protein loss, consistent with previous reports (Pradhan and Roy, 2012) [21]. Blood glucose concentrations remained within the normal physiological range across all groups, indicating that blood glucose levels are not directly influenced by renal dysfunction unless complicated by diabetes mellitus or severe stress conditions.

Overall, the hematological findings of the present study confirm that anemia and neutrophilia are prominent hematological features associated with renal dysfunction in dogs. The biochemical alterations observed reflect impaired excretory and metabolic functions of the kidneys. These findings are consistent with previous reports indicating that CKD in dogs is characterized by normocytic, normochromic anemia, azotemia, and mild changes in protein metabolism.

Conclusion

The present study demonstrated that dogs affected with renal failure exhibited marked hematological and biochemical alterations, reflecting impaired renal function and systemic effects of kidney damage. These changes emphasize the importance of routine evaluation of hematological and biochemical parameters as part of a comprehensive diagnostic approach for early detection, differentiation, and effective management of acute and chronic renal diseases in canine patients. Early identification through such diagnostic profiling can aid in timely therapeutic intervention, improved prognosis, and better clinical outcomes in affected dogs.

Conflict of Interest

Not available

Financial Support

Not available

Reference

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