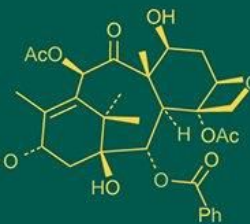
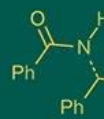


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Clinical and hematobiochemical profiling of canine renal failure

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Abstract

Renal failure in dogs is characterized by progressive deterioration of kidney function, leading to accumulation of nitrogenous waste products. The present study was conducted at the Department of Veterinary Clinical Complex, Veterinary Colleges at Bidar and Gadag from August 2023 to July 2025 to evaluate clinical, hematological, and biochemical alterations in dogs affected with renal failure. Dogs exhibiting signs such as anorexia, vomiting, oliguria/anuria, polyuria, polydipsia, weight loss, and poor body condition were included. Diagnosis was based on clinical examination, serum creatinine (>1.4 mg/dL), hematobiochemical analysis, and imaging techniques. Ten healthy dogs served as controls. The most common clinical findings were anorexia, lethargy, vomiting, dehydration and oliguria. Hematological evaluation revealed significant decreases in hemoglobin, packed cell volume, and total erythrocyte count, indicating anemia, along with significant leukocytosis. Biochemically, affected dogs showed markedly elevated blood urea nitrogen and serum creatinine, confirming impaired renal function. Serum phosphorus levels were significantly increased, while mild hypoalbuminemia was observed without significant changes in total protein. Although sodium and potassium levels showed statistical variation, they remained within normal physiological limits. A significant decrease in bicarbonate levels indicated metabolic acidosis. The study concludes that renal failure in dogs is associated with distinct clinical signs, anemia, leukocytosis, azotemia, hyperphosphatemia, hypoalbuminemia and metabolic acidosis. Comprehensive evaluation using clinical, hematological, and biochemical parameters is essential for accurate diagnosis and effective management of renal failure in dogs.

Keywords: Anemia, azotemia, hypoalbuminemia, hyperphosphatemia, metabolic acidosis

1. Introduction

Renal failure is deterioration of renal function leading to accumulation of nitrogenous wastes due to failure of kidneys to filter waste products adequately from blood (Labato, 2001) [33]. The second leading cause of non-accidental deaths in dogs was renal failure and the average age of onset of renal failure was over 6.9 years in dogs (Lewis *et al.*, 1987) [35]. The presumptive etiology of renal failure in dogs is mainly due to inflammatory causes, pyelonephritis, and ischemic causes and other or unknown (Dunaevich *et al.*, 2020) [15]. The commonest clinical outcomes of acute renal disease in dogs were gastrointestinal abnormalities, which included vomiting, oliguria and anuria, hematuria, halitosis, oral lesions and ulceration (Tufani *et al.*, 2015) [68]. In addition to this the chronic renal failure dogs had other clinical signs *viz.* polyuria and polydipsia, anemia, loss of body weight, lethargy and depression (Robinson *et al.*, 1989) [49]. Diagnosis of acute renal failure is usually based on the combination of complete history, clinical examination, and laboratory analysis and ultrasound examination. The minimum database for diagnosis of kidney affection includes hematobiochemical examination and imaging techniques. Therefore, this study was planned to evaluate the clinical and haemato-biochemical changes in the renal failure affected dogs.

2. Materials and Methods

The study was conducted at Department of Veterinary Clinical Complex, Veterinary College, Bidar and Gadag for a period of two years from August 2023 to July 2025. Dogs having signs such as Anorexia, selective or reduced appetite, anuria/oliguria, vomition, polyurea, polydipsia, gradual weight loss, poor hair coat, halitosis, apathy and poor body condition

(Ettinger and Feldmen, 2010) [18] Were selected as subjects for the study. A thorough general physical examination was performed clinical parameters such as physical condition, hydration status, colour of the mucous membrane, respiratory rate, heart rate and rectal temperature were documented in the clinical case file. Blood was collected from these dogs and the serum creatinine values were determined and the dogs having serum creatinine >1.4mg/dL were selected for the study group (n=30) and ten apparently healthy dogs with serum creatinine values of ≤ 1.4 mg/dl were selected as control group. Hematological studies were conducted using SMART-3DX Veterinary Hematology Analyzer (Manufactured by Byovet Medical & Technologies Private Limited, India) and biochemistry was carried out with Adiduoplus biochemistry analyzer (Manufactured by ADI Diagnostics Pvt. Ltd, India) through standard procedures described by the biochemical kit manufacturer. Blood urea nitrogen was estimated by Diacetyl monozyme method (Marsh *et al.*, 1957) [38], and is expressed in units of mg/dL. Serum creatinine was estimated by the modified Jaffe's alkaline picrate method (Folin and Wu, 1919) [21] and concentration of creatinine was expressed as mg/dL. Total plasma protein was estimated by modified biuret and bromo cresol green dye method (Dumas, 1975) [14] and expressed in g/dL. Albumin was estimated by Bromo cresol green end point assay method (Dumas, 1975) [14] and is expressed in g/dL. Potassium is estimated by end point method (Terri, 1958) [66] and is expressed in mEq/L. Microprocessor-controlled electrolyte system that uses ISE (Ion Selective Electrode) technology to make electrolyte measurements was used to measure serum sodium and potassium and the values were expressed as mEq/L. Bicarbonate was estimated as per the procedure described by Tietz (1964) [67]. The values were expressed in mEq/L. This study protocol was reviewed and approved by the Institutional Animal Ethical Committee, (IAEC) of Veterinary College, Bidar. The project proposal number is VCB/IAEC/VMD-16/2023-24. Statistical analysis was performed with GraphPad Prism 8.4 software (GraphPad, CA, USA). The data was presented by using bar diagrams and tables.

3. Results and Discussion

3.1 Clinical Manifestations

In the present study, the most important clinical signs observed in majority of the affected dogs were anorexia (83.33%), lethargy (80%), vomiting (66.67%), dehydration and Oliguria (30.00%), polyuria, and polydipsia (13.33%), pale/anaemic mucosa and gum and weight loss (10.00%), dental tartar and halitosis, malena, hypothermia and oral ulceration (6.67%), haematemesis, ascites, dyspnoea and seizures (3.33%) as enumerated in table 1 and Fig.1. These findings were in close agreement with those recorded by Cowgill (1995) [7], Behrend *et al.* (1996) [2], Vaden *et al.* (1997) [71], Haller (2002) [26] and Kralova *et al.* (2010) [31]. The uremia typically causes gastrointestinal (GI) signs including nausea, vomiting, diarrhea, anorexia, gastrointestinal and oral ulcerations, with glossitis, stomatitis and halitosis (Nivy *et al.*, 2021) [40]. Oliguria was observed in 30 per cent (n=9) clinical cases of the present study. The development of oliguria involves both hemodynamic and mechanical mechanisms. Hemodynamically, reduced blood volume (hypovolemia) and low blood pressure (hypotension) cause renal

vasoconstriction and poor perfusion (Finco *et al.*, 1979; Forrester and Little, 1994) [20, 22]. Mechanically, the nephrons may become blocked by thickened cellular casts or compressed by surrounding interstitial edema. These obstructions interfere with the passage of filtrate, ultimately lowering the glomerular filtration rate (Grauer and Lane, 1994; Schulman and Krawiec, 2000) [25, 54]. Polyuria and Polydipsia are due to glomerular hyper filtration and systemic hypertension (English, 1973 and Robinson *et al.*, 1989) [17, 49]. Anaemia and paleness of mucous membranes were recorded in 10 per cent (n=3) cases and it may be due to decreased production of erythropoietin factor from diseased kidneys, blood loss in the form of hematuria and decreased survival of RBC's due to uremic intoxication (Cowgill, 2003) [5]. Ascites could be due to diminished production of albumin, excretion of proteins through urine or sodium retention by diseases of kidney (Kaneko *et al.*, 2008) [29]. Severe metabolic acidosis leads to tachypnea and increased tidal respiration, decreased cardiac arrhythmias, peripheral arterial vasodilation and central vasoconstriction which can aggravate pulmonary oedma in dogs with renal failure (Shapiro, 1997) [57].

3.2 Physiological parameters

The Mean $\pm$ SE values of rectal temperature of healthy control group and renal failure dogs were 101.70 $\pm$ 0.20 and 101.14 $\pm$ 0.22 respectively. This observation suggested that body temperature was not affected but fluctuated within the normal physiological limits in renal failure dogs, which is in agreement with the findings of Elgazzar *et al.* (2021) [16]. Uremic patients exhibited hypothermia in comparison to nonuremic patients, and the hypothermia also indicated the severity of uremia (Kabatchnick *et al.*, 2016) [28]. In the present study there was significant increase in the heart rate in renal failure dogs (118.50 $\pm$ 7.62) compare to healthy dogs (82.80 $\pm$ 3.41). But the mean values of heart rate remained in the normal physiological range. Similarly, Sidhu (2015) [59], Singh (2017) [61] and Dogra (2018) [13] also observed normal mean heart rate in dogs suffering from renal failure. The elevated heart rate in renal failure dogs could be attributed to anemic and dehydration condition of the patients and the values were in the normal physiological range. This was in agreement with the findings of Elgazzar *et al.* (2021) [16]. The Mean $\pm$ SE values of respiration rate of renal failure dogs and healthy control dogs were 28.80 $\pm$ 1.10 and 26.90 $\pm$ 0.87 respectively. This is in agreement with the findings of Elgazzar *et al.* (2021) [16] and Sidhu (2015) [59]. Respiratory failure alters body buffering mechanism and leads to acute hypercapnia and then chronic hypercapnia causing increased renal excretion of acid. During chronic hypercapnia there is increased resorption of bicarbonate ions by renal tubules in order to maintain increased extracellular bicarbonate ions concentration (Simon and Greco, 1975) [60].

3.3 Hematology

The Mean $\pm$ SE values of hematobiochemical parameters are enumerated in table 2. The Mean $\pm$ SE values of hemoglobin, packed cell volume and total erythrocyte count in the healthy control group were 13.85 $\pm$ 0.37, 39.85 $\pm$ 1.50 and 7.12 $\pm$ 0.67 respectively, whereas the values were significantly decreased to 10.21 $\pm$ 1.02, 30.36 $\pm$ 3.45 and 4.19 $\pm$ 0.45 respectively in renal failure group of dogs. This was in agreement with Ganesh *et al.* (1981) [24], Vaden (2000) [70], Srivastava *et al.* (2011) [64], Devipriya *et al.*

(2018) [8], Sumit *et al.* (2018) [65] and Dunaevich *et al.* (2020) [15]. Anemia can be seen in ARF due to blood loss in haemorrhage, hemodilution or concurrent infection like leptospirosis that cause anemia and anemia is a feature in CRF due to either reduced erythropoietin secretion (Polzin *et al.*, 1995) [48] or decreased RBC life span, uremic inhibitors of erythropoiesis and external blood loss (Cowgill, 1995 and Osborne *et al.*, 1995) [7, 42, 44]. The Mean±SE value of Total leucocyte count (TLC) of renal failure dogs was significantly higher (15.86±1.76) compare to healthy control dogs (6.89±0.79). This was in agreement with the recordings of DiBartola *et al.* (1985) [11], Benjamin (1986) [3], Devipriya *et al.* (2018) [8] and Sumit *et al.* (2018) [65]. Several researchers have documented leukocytosis in animals suffering from renal failure, including Robison *et al.* (1989) [73], Ihle and Kostolich (1991) [27], Srivastava *et al.* (2011) [64], Kavitha *et al.* (2013) [30], Devipriya *et al.* (2018) [8], and Sumit *et al.* (2018) [65]. In contrast, Oburai *et al.* (2015) [41] found no significant differences in total or differential leukocyte counts between dogs with chronic renal failure (CRF) and healthy controls. The elevated TLC in the present study may be due to renal failure caused by leptospirosis, cystitis and inflammatory changes in kidney structures. In renal failure patients, leukocytosis typically reflects inflammatory processes within one or more organ systems. Specifically, when inflammation originates in the urinary tract, it usually involves the parenchymal tissue rather than the excretory pathways (Osborne and Finco, 1995) [19]. Furthermore, underlying systemic conditions that affect the urinary system—such as leptospirosis, bacterial endocarditis, and pyometra are frequently associated with leukocytosis (Osborne *et al.*, 1972; Adin and Cowgill, 2000) [43, 1]. In the present study no significant difference was noticed in platelet count in renal failure dogs (2.56±0.56) and healthy control dogs (3.87±0.54). The mean value of platelets was in accordance with Mann (2013) [37] and Singla *et al.* (2016) [62]. The observations with respect to platelets in the present study may be due to fewer cases of renal failure having the history of pre-existing Ehrlichiosis (n=6) and Babesiosis (n=3) infection and out of which only four cases were having mild thrombocytopenia.

3.4 Biochemistry

The Mean±SE values of biochemical parameters are enumerated in table 2. In the present study, the Mean±SE values of BUN and creatinine in the healthy group were 10.46±1.19 and 0.90±0.09 respectively, whereas they were significantly increased to 54.28±6.09 and 4.06±0.30 respectively in renal failure Group. Similar findings of the study were also observed by DiBartola *et al.* (1985) [11], Dighe *et al.* (1990) [12], Forterre *et al.* (2004) [23] and Sumit *et al.* (2018) [65]. Finco (1995) [19], DiBartola (2000) [10] and Lefebvre (2011) [34] reported that blood urea nitrogen (BUN) is synthesized in the liver from ammonium and bicarbonate and urea is excreted exclusively by the kidneys. Blood urea concentrations are influenced by factors such as protein catabolism, increased dietary protein intake and urine flow rate. demonstrated that elevated BUN levels are of clinical importance in the detection of renal impairment. Creatinine is produced in muscle through the spontaneous, non-enzymatic breakdown of phosphocreatine and is freely filtered by the glomerulus. Therefore, an increase in plasma creatinine concentration serves as a reliable indicator of impaired renal function and a reduction in glomerular

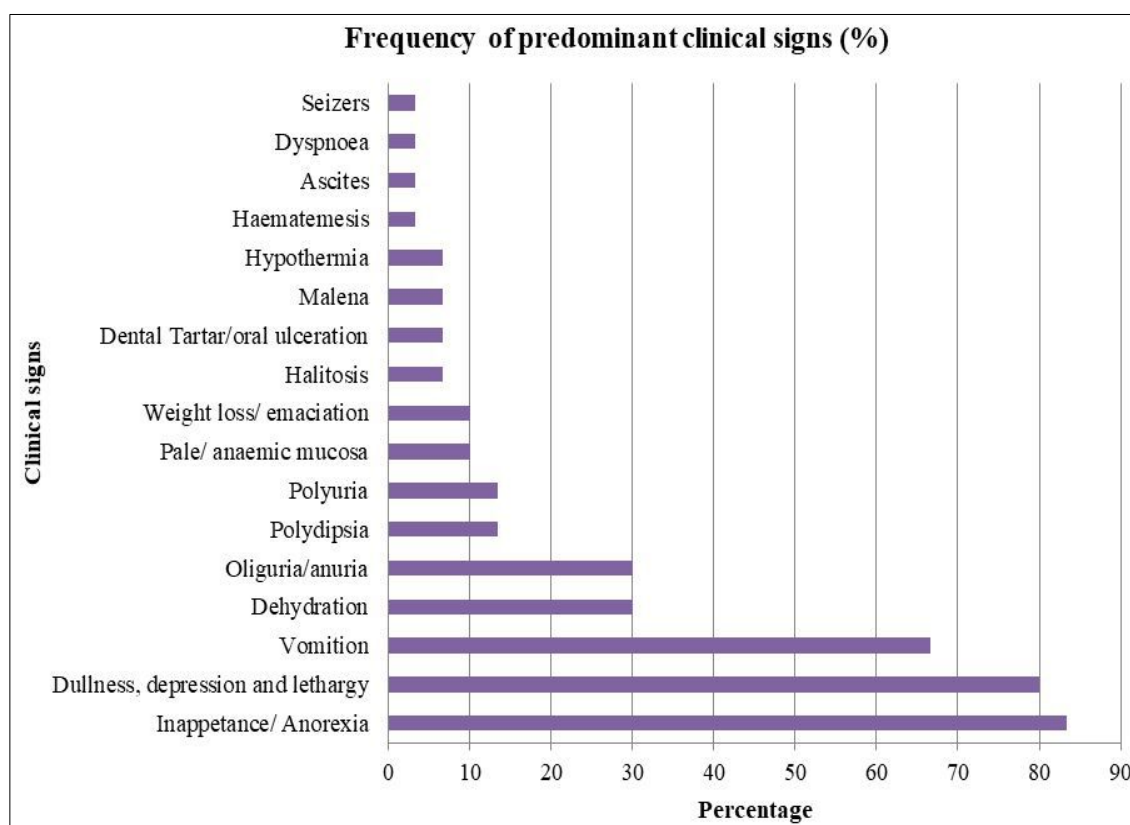
filtration rate (GFR) (DiBartola, 2000 and Haller, 2002) [10, 26], reflecting renal damage. The Mean±SE values of serum phosphorous of all the affected dogs were significantly increased in renal failure dogs (7.14±0.10) compared to healthy dogs (4.63±0.11). Similar findings were also observed by Ross (2011) [51], Polzin (2011) [46] and Sumit *et al.* (2018) [65]. Kidneys are the primary route of phosphorus excretion and declining kidney function results in phosphorus retention. Retention of excess phosphorus in the body can promote renal secondary hyperparathyroidism, mineralization of tissues and progression of CKD (Polzin, 2013) [47]. An unaltered change in the values of total protein (TP) in the renal failure dogs (5.98±0.19) compare to healthy control dogs (6.33±0.32) was observed in the present study whereas, a significant mild hypoalbuminemia was noticed in the renal failure dogs (2.50±0.11) compare to healthy control dogs (3.05±0.13). These findings were in close agreement with Seema (2007) [55] and Sumit *et al.* (2018) [65]. On contrary hypoproteinemia in renal failure dogs was observed by Srikanth and Satish (2014) [63] and Shruti *et al.* (2017) [58]. Hypoalbuminemia in kidney disease may occur due to urinary protein loss (Polzin, 2010) [45]. In the present study, the Mean±SE values of sodium and potassium in all the healthy control group were 138.60±0.56 and 3.92±0.47 respectively and they were significantly increased to 148.79±1.26 and 4.73±0.13 respectively in renal failure group. Although a statistically significant difference was observed in the mean sodium and potassium concentrations in between the two groups the mean sodium and potassium values in the present study remained within normal physiological limits in the affected dogs. These findings are consistent with the report of Schaer (1995) [53], who stated that serum sodium concentrations generally remain normal in patients with uremia. Neither hyponatremia nor hypernatremia was recorded during the present investigation. Nevertheless, disturbances in sodium balance may occur when there is an imbalance between body water and sodium, particularly in oliguric patients or following inappropriate fluid therapy. Similar observations have been reported by Rubin (1995) [52], Schaer (1995) [53], Cowgill and Francey (2005) [6] and Seema (2007) [55]. The Mean±SE values of bicarbonate in the renal failure and healthy dogs were 17.20±0.45 and 20.82±0.39 respectively, where a significant decrease in the bicarbonate values was recorded in renal failure group. These findings were in agreement with Chambers (1987) [4], Ross (1989) [50], McGrotty (2008) [39], Vijay *et al.* (2011) [72], Lippi *et al.* (2023) [36] and Segev *et al.* (2024) [56]. The metabolic acidosis in AKI results from increased acid generation, decreased bicarbonate reabsorption, and decreased acid secretion. High anion gap metabolic acidosis is also recognized with hyperlactatemia (associated with decreased tissue perfusion) in human and veterinary patients secondary to both diabetic ketoacidosis and toxin ingestion (*e.g.* ethylene glycol) (Ueda *et al.*, 2015) [69]. Metabolic acidosis can also be caused by loss of HCO₃-rich fluid from the body, addition of fixed acid to the body or its production by metabolism within the body or failure of renal excretion of fixed acid from the body. Loss of HCO₃-rich fluid usually occurs via the gastrointestinal tract (*e.g.*, small bowel diarrhea), but it also may occur via the kidneys (*e.g.*, carbonic anhydrase inhibitors, proximal renal tubular acidosis) (DiBartola, 2012) [9].

Table 1: Frequency of clinical signs in renal failure dogs

Sl. No.	Clinical sign	No. of renal failure dogs showing the respective clinical signs	Frequency of predominant clinical signs in Percentage
1	Inappetance/Anorexia	25	83.33
2	Dullness, depression and lethargy	24	80.00
3	Vomition	20	66.67
4	Dehydration	9	30.00
5	Oliguria/anuria	9	30.00
6	Polydipsia	4	13.33
7	Polyuria	4	13.33
8	Pale/anaemic mucosa	3	10.00
9	Weight loss/emaciation	3	10.00
10	Halitosis	2	6.67
11	Dental Tartar/oral ulceration	2	6.67
12	Malena	2	6.67
13	Hypothermia	2	6.67
14	Haematemesis	1	3.33
15	Ascites	1	3.33
16	Dyspnoea	1	3.33
17	Seizers	1	3.33

Table 2: Hematobiochemical parameters in healthy control and renal failure dogs

	Hematobiochemical parameters	Healty control (n=10)	Renal Failure dog (n=30)
1	Haemoglobin (g/dL)	13.85±0.37 ^a	10.21±1.02 ^b
2	PCV (%)	39.85±1.50 ^a	30.36±3.45 ^b
3	TEC(×10 ⁶ /μl)	7.12±0.67 ^a	4.19±0.45 ^b
4	TLC(×10 ³ /μl)	6.89±0.79 ^a	15.86±1.76 ^b
5	Platelets(×10 ⁵ /μL)	3.87±0.54 ^a	2.56±0.56 ^b
6	BUN (mg/dL)	10.46±1.19 ^a	54.28±6.09 ^b
7	Creatinine (mg/dL)	0.90±0.09 ^a	4.06±0.30 ^b
8	Phosphorous (mg/dL)	4.63±0.11 ^a	7.14±0.10 ^b
9	Total protein (g/dL)	6.33±0.32	5.98±0.19
10	Albumin (g/dL)	3.05±0.13 ^a	2.50±0.11 ^b
11	Sodium (mEq/L)	138.60±0.56 ^a	148.79±1.26 ^b
12	Potassium (mEq/L)	3.92±0.47 ^a	4.73±0.13 ^b
13	Bicarbonates (mmol/L)	20.82±0.39 ^a	17.20±0.45 ^b

**Fig. 1:** Frequency of predominant clinical signs in renal failure dogs

Conclusion

Renal failure in dogs is characterized by prominent clinical signs such as anorexia, vomiting, lethargy and altered urination patterns, along with significant hematobiochemical alterations. The study revealed anemia, leukocytosis, azotemia, hyperphosphatemia, mild hypoalbuminemia and decreased bicarbonate levels indicating metabolic acidosis. Physiological parameters largely remained within normal limits despite statistical variations. These findings emphasize that a combination of clinical evaluation and hematobiochemical profiling is essential for accurate diagnosis, assessment of disease severity and effective management of renal failure in dogs.

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