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Research Article

Journal of Veterinary and Animal Sciences
ISSN (Print): 0971-0701, (Online): 2582-0605
https://doi.org/10.51966/jvas.2026.57.2.217-224

Aetiological investigation of azotemia and oxidative profiling of postrenal azotemic cats in central Kerala#

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Citation: Keerthana, P.P., Manju, K. M., Madhavan, Unny. N., Sangeetha, S. Gireesh., Y. Ajith. & S. Anoop. (2026). Aetiological investigation of azotemia and oxidative profiling of postrenal azotemic cats in central Kerala. *Journal of Veterinary and Animal Sciences, 57(2),217-224*
https://doi.org/10.51966/jvas.2026.57.2.217-224

Received: 12.09.2025 Accepted: 14.11.2025 Published: 30.06.2026

Abstract

Azotemia in cats, characterised by elevated blood urea nitrogen and serum creatinine due to impaired renal clearance, is classified as prerenal, renal, or postrenal. It is a critical biochemical marker for early renal dysfunction and guides interventions such as fluid resuscitation, electrolyte correction, and urinary decompression. Oxidative stress, resulting from an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, plays a pivotal role in disease progression. The present study aimed to identify the etiology of azotemia and evaluate oxidative status in cats with postrenal azotemia. Cats presented to the University Veterinary Hospital, Kokkalai and Mannuthy, with clinical signs of azotemia were screened, and 50 cats with serum creatinine above 1.6 mg/dL were selected. Among the total cases, 60% were identified to be prerenal in origin and 40% were postrenal. The factors contributed to development of prerenal azotemia were haemoprotozoal diseases, gastrointestinal disorders and associated dehydration. Postrenal azotemia was due to obstructive uropathies. Haemato-biochemical analysis of azotemic cats revealed significant increase in total leucocyte, neutrophil, monocyte, eosinophil, erythrocyte, haemoglobin, creatinine, blood urea nitrogen and phosphorous level in diseased group compared with control. In the oxidative profile assessment, the mean total antioxidant capacity and glutathione peroxidase values were higher in diseased cats compared to controls. Early detection of oxidative imbalance may support timely therapeutic intervention and improved urinary tract management in feline patients.

Keywords: *Postrenal azotemia, total antioxidant capacity, glutathione peroxidase*

Kidney diseases are prevalent in companion animals, especially cats, and significantly contribute to clinical morbidity and mortality (Sparkes et al., 2016). Azotemia, characterised by elevated blood levels of nonprotein nitrogenous compounds may be manifested in prerenal and postrenal forms. Prerenal azotemia results from reduced renal perfusion, while postrenal azotemia arises due to obstructed urinary outflow commonly caused by urethral blockage, urolithiasis, or neoplasia and constitute a medical emergency in feline practice (Cooper and Scansen, 2020). This condition not only leads to nitrogenous waste accumulation and electrolyte imbalances but also predisposes cats to structural and functional renal damage, compromising urinary health (Lee and Drobatz, 2003).

#Part of MVSc thesis submitted to Kerala Veterinary and Animal Sciences University, Pookode, Wayanad, Kerala

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Oxidative stress, defined by an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, plays a pivotal role in renal pathology. Mitochondria generated ROS can damage cellular lipids, proteins and nucleic acids, leading to cell death and tissue injury (Majrashi et al., 2020). Renal tubular epithelial cells, rich in mitochondria, are particularly vulnerable to oxidative damage (Koyner et al., 2008; Daenen et al., 2019). The antioxidant defense system comprises of enzymatic and non-enzymatic components (Behzadian and Khoshvaghti, 2022). Glutathione peroxidase (GPx), a vital selenoenzyme, mitigates oxidative damage by reducing hydrogen peroxide and lipid hydroperoxides (Lubos et al., 2011). Total antioxidant capacity (TAC) offers a comprehensive measure of plasma's antioxidant potential, integrating both enzymatic and non enzymatic factors (Durak et al., 1999, Dounousi et al., 2006), data on their relevance in feline postrenal azotemia remain limited.

The present study investigates oxidative status in postrenal azotemic cats by measuring TAC and GPx activity, comparing results with healthy controls. These findings aim to elucidate the role of oxidative stress in feline postrenal azotemia and identify potential biomarkers for early diagnosis, prognosis, and therapeutic strategies.

Materials and methods

Collection of clinical data

Cats presented to UVH Kokkalai and TVCC Mannuthy, with clinical signs suggestive of azotemia *viz* vomiting, lethargy, diarrhoea and weight loss were screened in the present study. Comprehensive clinical history was taken and animals were subjected to detailed general clinical examination. Cats with serum creatinine above 1.6 mg/dL were subjected to detailed etiopathological investigation to identify the etiology of azotemia and the cats diagnosed with postrenal azotemia were subjected to oxidative profiling. Six clinically healthy cats served as controls, irrespective of age, breed, and gender.

Sample collection and processing

Blood samples were collected aseptically from the cephalic or saphenous vein (Malik et al., 2011). Complete blood count, serum biochemistry parameters *viz* serum creatinine, blood urea nitrogen and phosphorus were estimated. Plasma was separated from EDTA vial blood samples of cats affected with postrenal azotemia and stored at –20 °C until further analysis of oxidative status (Quintavalla et al., 2024).

Diagnostic imaging techniques

Diagnostic imaging techniques *viz* lateral abdominal radiography and ultrasonography were conducted in the selected cats.

Estimation of antioxidant status

Oxidative profiling of cats affected with postrenal azotemia was carried out. Total antioxidant capacity (TAC) was estimated using the ferric reducing antioxidant power (FRAP) assay. At low pH, reduction of ferric tripyridyl triazine (Fe^{3+}–TPTZ) to its ferrous form generated an intense blue color, and the change in absorbance was measured at 593 nm using a Perkin Elmer Lambda UV-VIS spectrophotometer (Gupta et al., 2021). The change in absorbance was directly proportional to the reducing power of electron-donating antioxidants present in the plasma (Rubio et al., 2017).

Glutathione peroxidase (GPx) activity was measured by a conventional biochemical method in which GPx catalysed the oxidation of glutathione by hydrogen peroxide (Ahmed et al., 2021). The oxidized glutathione was reduced by glutathione reductase in the presence of Nicotinamide adenine dinucleotide phosphate (NADPH) and the oxidation of NADPH to NADP was monitored spectrophotometrically at 340 nm (Ramadhani et al., 2021) using the same instrument.

Statistical analysis

Data were analysed using SPSS software version 24.0. Results were expressed as mean ± standard error (SE). Independent t-test, Chi – square test and Mann-Whitney test were used to compare means between diseased and control groups, and statistical significance was considered at $p < 0.05$ and $p < 0.01$ levels.

Results and discussion

Among the 262 cats which were presented with clinical signs suggestive of azotemia from January 2025 to June 2025, 50 cats (19.08 per cent) were found to have serum creatinine value above 1.6 mg/dL.

Of the fifty cats studied, 30 cats (60%) were diagnosed with prerenal azotemia and 20 cats (40%) with postrenal azotemia. Prerenal azotemia was identified mainly secondary to haemoprotozoal diseases (40%). The major haemoprotozoal diseases identified were *Mycoplasmosis* and *Cytauxzoonosis*. Rest 20 per cent of the prerenal azotemic cats were identified with gastrointestinal disorders and associated dehydration. Postrenal azotemia developed due to obstructive uropathies in all the cats.

Demographic characteristics of the diseased cats

Demographic characteristics *viz* breed, gender, age and body condition score are represented in Table 1. The highest occurrence of prerenal azotemia in domestic short hair cats may be due to their larger population but not a true breed predisposition (Williams et al., 2024), whereas the higher occurrence of lower urinary tract disease in Persian breed of cats possibly might be due

Table 1. Statistical analysis of demographic parameters

Parameter	Diseased group (n=50)		Control group (n=6)		Test statistics		Test outcome	
	T_1 (n=30)	T_2 (n=20)	T_1 (n=30)	T_2 (n=20)	T_1	T_2	P_1	P_2
Breed	Persian: 46.67% DSH: 53.3%	Persian: 75% DSH: 15% Himalayan: 5% Crossbred: 5%	Persian: 83.3% DSH: 16.7%	Persian: 83.3% DSH: 16.7% Himalayan: 0% Crossbred: 0%	χ^2 value :0.089^{ns}	χ^2 value :0.650^{ns}	0.148	0.885
Gender	Male: 46.7% Female: 53.33%	Male: 90% Female: 10%	Male: 83.33% Female: 16.67%	Male: 83.33% Female: 16.67%	χ^2 value :1.426^{ns}	χ^2 value :0.201^{ns}	0.232	0.654
Age	30.27 ± 2.54	27.68 ± 3.06	22.40 ± 4.05	22.40 ± 4.05	t value: 1.643^{ns}	t value: 0.871^{ns}	0.133	0.392
Body condition score	17.18	12.98	15.25	15.25	U value: 70.50 Z value: 0.828^{ns}	U value: 49.50 Z value: 0.695^{ns}	0.408	0.487

T_1: Prerenal azotemia P_1 : Test outcome for Prerenal azotemia

T_2: Postrenal azotemia P_2 : Test outcome for Postrenal azotemia

to their genetic and metabolic tendencies to urolithiasis (Lekcharoensuk et al., 2001). Longer and narrow urethra might be the reason which had predisposed male animals to obstruction and hence postrenal azotemia (Abdel-Saeed et al., 2021). Most of the cats affected with both prerenal as well as postrenal azotemia were found to be within the age group of young adult, which might be due to their higher metabolic rate, vulnerable to dietary mineral imbalances or stress related urinary changes (Longstaff et al., 2017). The mean ± SE bodyweight of cats affected with prerenal azotemia was 2.79 ± 0.13 kg and postrenal azotemia was 3.71 ± 0.18 kg and these findings were contradictory to the findings of Pusoonthornthum et al. (2012) where they have reported that obese cats had higher incidence of developing postrenal azotemia. The findings reported in BCS were in contradictory with the findings of Teng et al. (2018) where they have reported higher occurrence of lower urinary tract diseases in cats with a higher BCS score.

No relationship was observed between the type of diet and development of prerenal azotemia, but cats with gastrointestinal disorders and associated dehydration with a sudden diet change had higher occurence. Highest occurrence of postrenal azotemia was reported in cats fed solely on a dry diet (75 per cent) (Piyarungsri et al., 2023) followed by a combination of both dry and wet (homemade) food (20 per cent) and wet food (5 per cent) as shown in Table 2.

Seventy per cent of prerenal cases occurred during the pre-monsoon summer, while nine cases (30%) were observed in winter and no cases were reported during

Table 2. Distribution of diet types in cats with postrenal azotemia (n= 20)

Type of diet	Diseased group (n=20)	Control group (n=6)	X^2 - value	p-value
Dry food	75.0%	50.0%	2.260^{ns}	0.323
Wet food	5.0%	0.0%		
Combination	20.0%	50.0%		
Total	100%	100%		

the monsoon or post-monsoon seasons of Koppen climate classification (Peel et al., 2007). Majority of the postrenal cases occurred in pre-monsoon summer which was contradictory to the findings of Martyniv, 2024. No postrenal cases were reported during the post-monsoon months, which is contradictory to the findings of Eggertsdottir et al. (2007) who reported highest occurrence during cooler and drier seasons and lowest during warmest summer months. These findings indicate that the pre-monsoon hot season poses the highest risk for both prerenal and postrenal conditions in cats. Overall, seasonal variation in renal cases appears to be influenced by temperature and precipitation patterns typical of the tropical monsoon climate.

Clinical signs

The most common clinical signs observed in prerenal azotemia were hyporexia to anorexia in all the cases, followed by lethargy exhibited in 95 per cent and 75 percent of the cases were having vomiting. In postrenal azotemic cats major findings included licking of the perineal region (100 per cent), abdominal distension (100

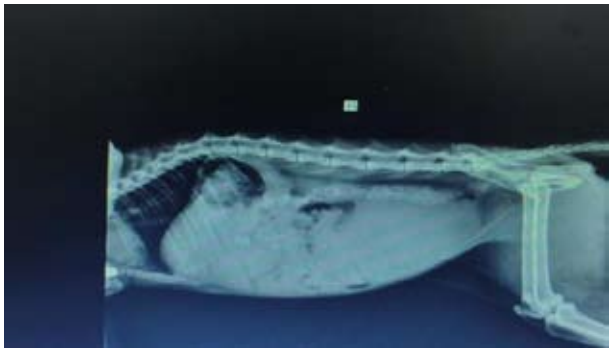

Fig. 1. Urinary bladder distention and faecal stasis in a cat affected with postrenal azotemia

per cent) (Jerabkova et al., 2024), anorexia (85 per cent), vomiting (75 per cent), anuria (60 per cent), dribbling of urine (40 per cent), stranguria (40 per cent) (Nithin et al., 2023), haematuria (40 per cent), hyporexia (15 per cent) and diarrhoea (15 per cent). Laboratory investigations comprised serum biochemistry, haematology, while radiography and ultrasonography were employed to confirm obstructive uropathies (Freitas et al., 2012). Cases were differentiated into prerenal or postrenal azotemia based on the presence of urinary obstruction and characteristic clinical findings.

Haemato biochemical analysis

The haematological values of prerenal azotemia revealed a significant elevation in total leucocyte count in the diseased group than control group. In the haemogram, the total erythrocyte count and the haemoglobin level of the diseased group was higher when compared to control group but not significant. Other major findings were the diseased group had a significant reduction in mean corpuscular volume (MCV), mean corpuscular haemoglobin concentration (MCHC) and platelet count when compared with control group enlisted in Table 3.

In cats with postrenal azotemia, the diseased group revealed a striking leukocytosis, driven predominantly by neutrophilia and a marked lymphopenia. This classic stress leukogram suggests acute inflammatory or infectious process (Cagnasso et al., 2022) with systemic stress mediated lymphocyte suppression. Monocyte counts dipped slightly, while eosinophils

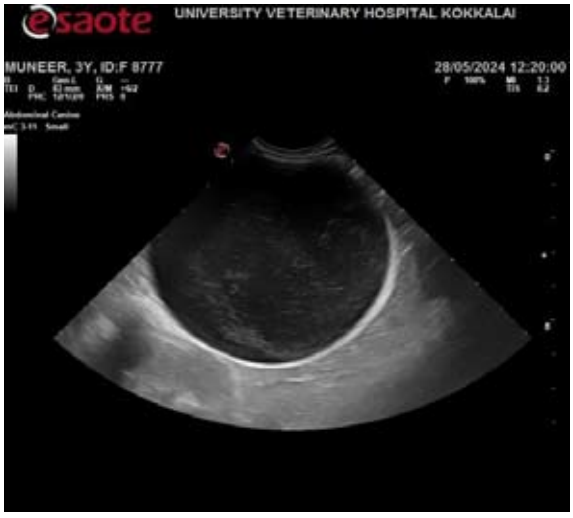

Fig. 2. Distended urinary bladder with hyperechoic contents inside

Fig. 3. Distended urinary bladder with blood clots inside

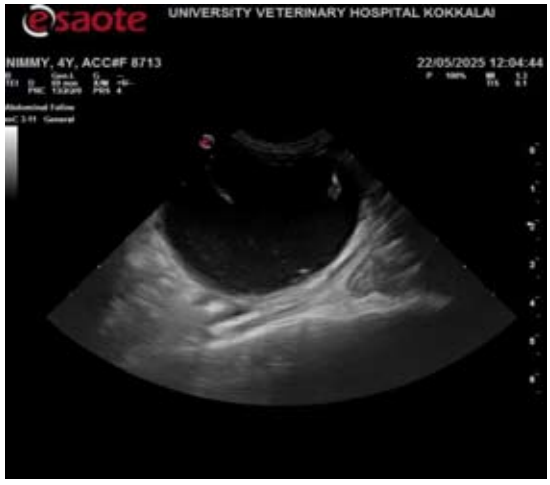

Fig. 4. Urethral distention secondary to urethral obstruction

Fig. 5. Perirenal effusion in a cat

Table 3. Comparison of haematological parameters in the prerenal diseased and control group (n= 30)

Parameters	Diseased group (n = 30)	Control group (n = 6)	t - value	p - value
Total leucocyte count (x 10^6/µL)	15.54 ± 0.73	11.38 ± 1.84	**2.264***	0.030
Lymphocyte (%)	20.01 ± 0.67	37.20 ± 0.71	**11.02****	0.000
Monocyte (%)	7.12 ± 0.14	5.33 ± 0.27	**5.21****	0.000
Neutrophil (%)	65.75 ± 0.81	50.12 ± 1.49	**9.16****	0.000
Eosinophil (%)	6.42 ± 0.11	5.45 ± 0.85	1.12	0.312
Basophil (%)	0.67 ± 0.06	1.87 ± 0.16	**7.019****	0.000
Total erythrocyte count (x 10^3/µL)	8.83 ± 0.38	7.78 ± 0.70	1.147	0.260
Haemoglobin (g/dL)	9.44 ± 0.40	10.48 ± 0.80	1.068	0.293
HCT (%)	26.92 ± 1.16	37.32 ± 2.60	**3.64****	0.001
MCV (fL)	30.85 ± 0.59	48.51 ± 1.95	**11.17****	0.000
MCHC (g/dL)	29.61 ± 0.46	33.50 ± 1.13	**3.347****	0.002
Platelet (x 10^3/µL)	52.16 ± 4.59	199.5 ± 32.68	**4.464****	0.006

Table 4. Comparison of haematological parameters in the postrenal diseased group and control group (n = 20)

Parameters	Diseased group(n=20)	Control group(n=6)	t - value	p- value
Total leucocyte count (x 10^3/µL)	24.37 ± 2.11	11.38 ± 1.84	**3.214****	0.004
Lymphocyte (%)	5.86 ± 0.86	37.2 ± 0.71	**27.937****	0.000
Monocyte (%)	3.41 ± 0.36	5.33 ± 0.27	**4.238****	0.000
Neutrophil (%)	88.37 ± 1.26	50.12 ± 1.49	**15.441****	0.000
Eosinophil (%)	2.10 ± 0.45	5.45 ± 0.85	**3.494****	0.002
Basophil (%)	0.24 ± 0.04	1.87 ± 0.16	**9.711****	0.000
Total erythrocyte count (x10^6/µL)	9.22 ± 0.43	7.78 ± 0.70	1.621^{ns}	0.118
Haemoglobin (g/dL)	13.32 ± 0.60	10.48 ± 0.80	**2.375***	0.026
HCT (%)	42.59 ± 1.66	37.32 ± 2.60	1.70	0.120
MCV (Fl)	44.81 ± 2.59	48.51 ± 1.95	0.754^{ns}	0.458
MCHC (g/dL)	31.50 ± 0.48	33.50 ± 1.13	1.869^{ns}	0.074
Platelet count (x10^3/µL)	183.75 ± 33.88	199.50 ± 32.68	0.242^{ns}	0.811

Table 5. Comparison of serum biochemical parameters in the prerenal diseased group and control group (n = 30)

Parameters	Diseased group(n=30)	Control group(n=6)	t - value	p- value
Creatinine (mg/ dL)	2.33 ± 0.13	1.10 ± 0.11	**3.94****	0.000

Table 6. Comparison of serum biochemical parameters in the postrenal diseased group and control group (n = 20)

Parameters	Diseased group(n=20)	Control group(n=6)	t - value	p- value
BUN (mg/dL)	70.89 ± 12.43	11.88 ± 0.87	**4.733****	0.00
Creatinine (mg/dL)	8.94 ± 1.62	1.10 ± 0.11	**4.820****	0.00
Phosphorous (mg/dL)	12.33 ± 0.76	4.81 ± 0.70	**7.228****	0.00

Table 7. Comparison of oxidative stress parameters in the postrenal azotemic cats and control group (n = 20)

Parameters	Diseased group (n = 20)	Control group (n = 6)	t - value	p - value
Total antioxidant capacity (TAC) (µmol/L)	995.94 ± 224.07	653.49 ± 105.99	0.818^{ns}	0.421
Glutathione peroxidase activity (GPX) (IU/L)	9.90 ± 1.26	12.83 ± 2.26	1.115^{ns}	0.26

and basophils were significantly reduced. In contrast, erythrocyte indices painted a subtler picture. Although total erythrocyte count and haematocrit were elevated in diseased cats, the differences weren't statistically significant, revealing haemoconcentration rather than true erythrocytosis. Notably, haemoglobin levels were significantly higher, potentially reflecting dehydration or compensatory erythropoiesis. Mean corpuscular volume and mean corpuscular haemoglobin concentration trends leaned toward microcytosis and hypochromia, though not significantly suggesting early or subclinical shifts in red cell morphology. Platelet counts remained within normal limits, ruling out thrombocytopenia as a confounding factor enlisted in Table 4.

Cats in the prerenal azotemic group exhibited dramatically elevated levels of creatinine when compared to control group as listed in Table 5 (Fonseca et al., 2022) whereas in case of postrenal azotemia, cats showed elevated levels of serum creatinine, blood urea nitrogen as well as phosphorous in diseased group when compared to control group as enlisted in Table 6. The findings underscored the severe compromise in renal excretory function, consistent with prerenal as well as postrenal azotemia (Acierno, 2022).

Radiographic findings

Lateral abdominal radiographs of all the cats affected with postrenal azotemia revealed marked urinary bladder distention and faecal stasis Figure 1.

Ultrasonographic findings

All the cats affected with postrenal azotemia had distended bladder (100 per cent). Hyperechoic structures were observed in all cases within the bladder which included crystalline deposits (Fig. 2), cellular aggregates and blood clots (Fig. 3) these findings were in accordance with Debruyn et al. (2012). Cats with urethral plugs were found to have thickened bladder wall (proliferative changes) and distended urethra (Nevins et al., 2015) (Fig. 4). Other major findings included altered echogenicity, cortical thickening, reduced cortico-medullary differentiation, peri-renal fluid accumulation (Fig. 5), pelviectasis and hyperechoic medullary rim signs and these findings were in accordance with Griffin (2020).

Oxidative profile

In this study, cats with postrenal azotemia showed elevated TAC and lower GPx activity compared to healthy controls, yet neither difference reached statistical significance ($p > 0.05$) as enlisted in Table 7. The elevated TAC may reflect a compensatory surge in non enzymatic antioxidants mobilised in response to oxidative stress (Sharma et al., 2022), as previously observed in acute renal insults (Kumar et al., 2020). Conversely, the reduced GPx activity suggests early enzymatic exhaustion or selenium-dependent downregulation, consistent with findings in obstructive uropathy models (Sugimoto et al., 2023). The lack of significance could stem from sample size limitations or temporal variability in oxidative marker expression. These results underscore the need for longitudinal profiling and larger cohorts to clarify the diagnostic and prognostic utility of oxidative biomarkers in feline urinary tract disease.

Conclusion

Azotemia in cats is characterised by elevated nitrogenous waste in the blood due to impaired renal clearance. This study investigated its causes and oxidative status, focusing on postrenal cases. Prerenal azotemia was mainly linked to haemoprotozoal and gastrointestinal disorders with dehydration, while postrenal azotemia resulted from urinary obstruction. Affected cats showed signs such as anorexia, vomiting and abdominal distension. Haematobiochemical findings indicated inflammation and impaired renal function. Oxidative profiling revealed increased total antioxidant capacity and reduced glutathione peroxidase activity, suggesting oxidative stress involvement in disease progression. Although not statistically significant, these changes reflect compensatory antioxidant responses. The study highlights oxidative imbalance as an important factor in feline azotemia and the need for early detection and management.

Acknowledgement

The authors of this paper are thankful to the Head of Teaching Veterinary Clinical Complex and Department of Veterinary Clinical Medicine, Ethics and Jurisprudence, Director of Clinics and the Dean, College of Veterinary and Animal Sciences, Mannuthy for providing the facilities for the conduct of current study.

Conflict of interest

The authors declare that they have no conflict of interest.

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