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Gastrointestinal and Hematobiochemical Effects of Elevated BUN with Renal Dysfunction in a Male Cat: A Case Report

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ABSTRACT

A two-year-old neutered tom, named Slavic, was presented with a history of vomiting, diarrhea, and dehydration. Clinical evaluation revealed elevated BUN, creatinine, phosphorus, and glucose levels alongside anemia and lymphopenia. A comprehensive diagnostic approach, including hematology, biochemistry, urinalysis, and radiography, confirmed azotemia and renal insufficiency. Gastrointestinal indications were attributed to uremia, which led to dehydration. Treatment protocol for seven days comprising intravenous (IV) fluids, antibiotics, gastro-protectants and nutritional support that partially stabilized the male cat. This case correlates with elevated BUN and gastrointestinal symptoms in feline renal diseases. A favorable prognosis is possible with timely diagnosis, supportive measures and nutritional concerns although continued monitoring and long-term management and observation is essential.

Keywords: Feline, BUN, Renal Dysfunction, Diarrhea, Azotemia, Gastrointestinal

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Introduction

Blood urea nitrogen (BUN) reflects the amount of nitrogen present in blood in the form of urea, a metabolic waste product resulting from the hepatic breakdown of proteins through the urea cycle [1]. Urea is generated in the liver and is then excreted by the kidneys. The BUN level is frequently used as a clinical marker of kidney function, as the kidneys play a primary role in filtering and excreting urea from the body [2].

In the gastrointestinal tract (GIT), BUN itself doesn't have a direct impact. As an alternative, the concentration of BUN in the blood is influenced by various factors associated with digestive physiology and protein metabolism, including dietary protein intake, hepatic urea cycle function, and hydration status [3]. When proteins are broken down during digestion, the amino acids are absorbed and moved to the liver, where they undergo a procedure called deamination [4]. During deamination, nitrogen is removed from amino acids and converted into ammonia. In the liver, this ammonia is detoxified through the urea cycle by combining with carbon dioxide to form urea, which is then transported to the kidneys for excretion [5].

Elevated BUN levels, known as azotemia, can result from impaired kidney function, dehydration, or increased protein breakdown. Elevated blood urea nitrogen (BUN) levels may be observed in cases of renal insufficiency, where impaired glomerular filtration leads to reduced urea excretion. When accompanied by diarrhea, the resulting dehydration can exacerbate azotemia, compounding the elevation of BUN [6]. The imbalance in nitrogen elimination can lead to higher concentrations of urea in the blood, potentially contributing to gastrointestinal disturbances. Additionally, dehydration often marked by elevated blood urea nitrogen (BUN) levels can contribute to changes in gastrointestinal motility and mucosal function, potentially leading to symptoms such as vomiting or diarrhea. It's important to note that while elevated BUN alone does not directly cause diarrhea, it may be indicative of an underlying condition affecting both renal function and gastrointestinal health [7].

The elevated bilirubin concentration, known as hyperbilirubinemia, can be attributed to various underlying factors within the context of hepatic and hematologic processes [8]. Liver diseases, including hepatitis or cirrhosis, may hinder hepatic conjugation of bilirubin, leading to its accumulation in the bloodstream [9].

In a male cat, normal blood urea nitrogen (BUN) levels typically range from 15 to 30 mg/dL. However, it's important to note that reference ranges can vary slightly between laboratories depending on methodology and instrumentation [10]. Abnormal blood urea nitrogen (BUN) levels can be indicative of several underlying health conditions. Elevated BUN levels—referred to as azotemia—may arise from impaired kidney function, dehydration, or increased protein catabolism. Conversely, low BUN levels may occur in association with liver disease, malnutrition, or overhydration [2].

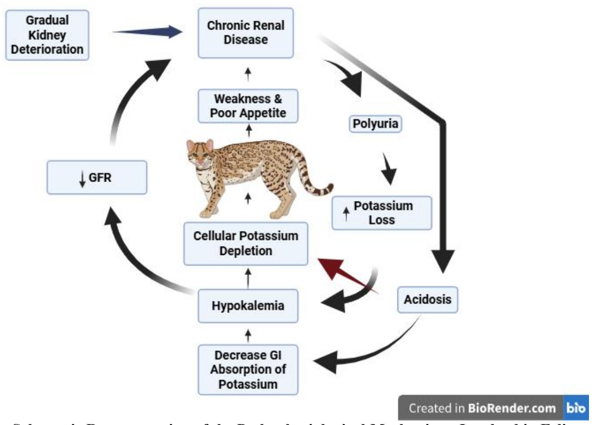

Fig. 1: Schematic Representation of the Pathophysiological Mechanisms Involved in Feline Chronic Kidney Disease

Case Presentation

A one-year-old, neutered male cat (Slavic) was brought to the clinic with a history of vomiting, diarrhea, dullness and emaciation for the last seven days. Upon physical examination, there was severe dehydration leading to sunken eyes, dry buccal and mucociliary membranes, increased skin tenting, and lethargy. Several hematological and biochemical test regimens were performed, indicating prominent deviations from normal, which showed that the male cat was experiencing discomfort. The aseptic blood samples were collected via jugular vein using sterile vacutainers. And performed a complete blood count (CBC). The findings showed low values of mean hemoglobin concentration and corpuscular volume, white blood cells (lymphocytes), red blood cells (hemoglobin) and platelets were low as compared to normal values which indicated anemia. Further analysis involved evaluation of hepatic enzymes and renal system profile of Slavic. The laboratory test results of Slavic were showing high albumin, severely suppressed alanine aminotransferase (ALT), high creatinine, high blood urea nitrogen (BUN), and significantly hyperproteinaemia. These findings collectively indicated renal and hepatic damage. The faecal examination revealed blackish-colored faeces, indicating gastrointestinal and kidney problems. Urine examination indicated the whitish colour of the urine which indicated kidney stones. Radiographic examination was performed to check the internal organs' size, appearance and abnormalities. The cumulative findings prompted a continuous monitoring approach, involving CBC and protein biochemistry tests over three days to gain comprehensive insights into Slavic's evolving health status.

Table 1: Complete blood count profile of the blood of cat (Dragon Animal Care Center Lab 2023)

Parameters	Test Results (Day 1st)	Test Results (Day 3rd)	Test Results (Day 7th)	Reference Ranges
WBC	12.36 x 10^4 ul	16.59 x 10^4 ul	3.57 x 10^4 ul	5.50-19.50
LYM	1.46 x 10^4 ul	7.43 x 10^4 ul	0.64 x 10^4 ul	1.50-7.00
MON	0.54 x 10^4 ul	0.10 x 10^4 ul	0.23 x 10^4 ul	0.00-1.50
NEU	10.21 x 10^4 ul	9.02 x 10^4 ul	2.66 x 10^4 ul	2.50-14.00
EOS	0.14 x 10^4 ul	0.04 x 10^4 ul	0.04 x 10^4 ul	0.00-1.00
LYM%	0.01 x 10^4 ul	44.8%	17.9%	0.00-0.20
MON%	11.8 %	0.6 %	6.4 %	0.0-100
NEU%	4.4 %	54.4 %	74.5 %	0.0-100
EOS%	82.6 %	0.2 %	1.2 %	0.0-100
BAS%	1.1 %	0.0 %	0.0 %	0.0-100
RBC	8.07 x 10^6 ul	5.60 x 10^6 ul	4.28 x 10^6 ul	5.00-10.00
HGB	12.3 g/dl	8.0 g/dl	6.2 g/dl	8.0-15.0
HCT	38.21 %	27.07 %	21.75 %	24.00-45.00
MCV	47 fl	48 fl	51 fl	39-55
MCH	15.2 pg	14.4 pg	14.5 pg	12.5-17.5
MCHC	32.1 g/dl	29.7 g/dl	28.6 g/dl	30.0-36.0
PLT	320 x 10^4/ ul	320 x 10^4/ ul	138 x 10^4 / ul	300-800
MPV	10.4 fl	9.7 fl	11.7 fl	12.0-17.0
PCT	0.33%	0.31%	0.16%	24-45%

Table 2: Biochemistry profile of the blood of cat (Dragon Animal Care Center Lab 2023)

Parameters	Test Results (Day 1st)	Test Results (Day 3rd)	Test Results (Day 7th)	Reference Ranges
ALB	5.6*	5.1*	3.9	2.2-4.4 g/dl
ALP	21	23	17	10-90 u/l
ALT	19*	66	39	20-100 u/l
AMY	1873*	1871*	684	300-1100 u/l
TBIL	0.3	0.4	0.4	0.1-0.6 mg/dl
BUN	> 180*	105*	57*	10-30 mg/dl
CA	11.9*	12.0*	9.6*	8.0-11.8 mg/dl
PHOS	16.1*	7.6*	5.8	3.4-8.5 mg/dl
CRE	5.5*	3.7*	2.7*	0.3-2.1 mg/dl
GLU	158*	135	144	70-150 mg/dl
NA+	153	153	151	142-164 mmol/dl
K+	3.6*	2.7*	3.3*	3.7-5.8 mmol/dl
TP	9.6*	7.4	6.2	5.4-8.2 g/dl
GLOB	4.0	2.4	2.3	1.5-5.7 g/dl

Treatment Plan

For managing elevated BUN in the subject, treatment involved fluid therapy via constant infusion of Ringer's lactate solution at a rate of 10ml/hr for restoration of hydration status within 24 hours. Gentamycin @0.16mg/kg was gently administered to remedy renal discomfort, thereby inducing analgesia. To prevent the development of bacterial infections, antimicrobial therapy via Penicillin @8-10mg/kg was given as an additional antibiotic in order to further prevent the development of bacterial infections. Elevated BUN Levels predispose the gut and stomach to subsequent inflammation and ulcers; therefore, it is essential to protect the stomach and gut to minimize such risk. This was achieved through the synergistic effect of metoclopramide @1mg/kg and Ranitidine 2.5mg/kg, which effectively reduces the rancidity.

The post-hospitalization plan for seven-day boarding included providing a supplemental nutrient diet in the form of (Happy Cat Kidney) diet, which is a custom diet specifically formulated to maintain renal health. This program was to be followed over six months. For normalizing potassium levels, Syp-Kaminix was administered orally; this medication stabilizes ionic disequilibria attributed to elevated levels of phosphorus, which is a common consequence of renal failure.

Discussion/Findings

Considering the CBC report, which indicated blood urea nitrogen levels higher than the normal range, our subject (Salvic) presented clinical signs such as diarrhea, vomiting, and severe systemic exertion. The laboratory investigation evidenced that BUN is a biomarker for nitrogenous waste accumulation, which directly results in gastrointestinal pathology. The pathophysiology of such metabolic disorder comprises gastric mucosal induction of uremia and general toxicity. These disturbances are manifested as nausea, vomiting, and melena in cats predisposed to CKD, chronic kidney disease [11,12].

Upper gastrointestinal bleeding was evident from unusual blackish smelly discharge, conformity with similar implications such as those of gastrointestinal ulceration and mucosal hemorrhage. These studies prove that vulnerability of the mucosal layer and platelets dysfunction encourage gastrointestinal bleeding, especially when accompanied by a lack of protective factors, and consequently lead to an increase in BUN/creatinine in cats [6,13]. Occult blood losses over time in the cat were indicated by a reduction in hematocrit and hemoglobin parameters [14,15]. Hypokalemia, specifically has been associated with decreased intestinal peristalsis and anorexia in feline CKD patients [16].

The treatment protocol was Ringer's Lactate, dexamethasone, ranitidine and metoclopramide, given for azotemic cats with suspected GI bleeding requiring individualized acid suppression [17].

Post-hospitalization management with a nutritional perspective involving a renal-specific diet such as "Happy Cat Kidney" was in congruence with best practices for dietary phosphorus restriction and protein modulation, which have been verified to prolong persistence and reduce GI complications in azotemic cats [18].

Gastrointestinal signs in azotemic cats can often mimic primary GI disorders; histopathological studies have shown that up to 60% of such cases are diagnosed falsely when there are renal disorders [19].

Conclusion

In summary, elevated BUN levels in cats may manifest not only as markers of renal compromise but also contribute to secondary gastrointestinal symptoms, possibly through uremia-related mucosal effects or dehydration. A detailed diagnostic approach and comprehensive supportive therapy, including dietary modifications, are crucial in managing such cases. Early intervention and continuous follow-up remain essential to improving long-term outcomes in feline patients with renal dysfunction.

References

[1] He Z, Lin Z, Yan Y, Wang J, Zhang S & Zheng B. 2025. Curcumin and selenium synergistically mitigate oxidative stress in white-feathered broilers. Frontiers in Veterinary Science, 12: 1650011.

[2] Pereira AF, Jota Baptista C & Faustino-Rocha A. 2025. Renal biomarkers in companion animals—A review. Animals, 15(6): 818.

[3] Xu Y, Li Z, Moraes LE, Shen J, Yu Z & Zhu W. 2019. Effects of incremental urea supplementation on rumen fermentation, nutrient digestion, plasma metabolites, and growth performance in fattening lambs. Animals, 9(9): 652.

[4] Saha SK, Pathak NN, Saha SK & Pathak NN. 2021. Digestion, Absorption and Metabolism of Nutrients. Fundamentals of Animal Nutrition, 219–246.

[5] Wang L, Li J, Wang B, Yin X & Wei J. 2024. Progress in modeling avian hyperuricemia and gout. Biomedical Reports, 20(2): Article 1879.

[6] Langston C & Eatroff A. 2015. Acute kidney injury. In August's Consultations in Feline Internal Medicine.

[7] Segev G, Foster JD, Francey T & Langston CE. 2024. International Renal Interest Society Best Practice Consensus Guidelines for Intermittent Hemodialysis in Dogs and Cats. The Veterinary Journal.

[8] Richter KP. 2009. Diseases of the liver and hepatobiliary system. In SJ Ettinger & EC Feldman (Eds.), Textbook of Veterinary Internal Medicine (7th ed.). WB Saunders.

[9] De Silva AP, Nuwanshika N, Gunatilake D & Samarasinghe R. 2025. Gilbert's syndrome: The good, the bad and the ugly. World Journal of Hepatology, 17(1): 12–26.

[10] International Renal Interest Society. 2023. IRIS Guidelines – Diagnosis and treatment of renal disease in cats and dogs.

[11] Gianella P, Pietra M, Crisi PE & Bergamini PF. 2017. Evaluation of clinicopathological features in cats with chronic gastrointestinal signs. Journal of Veterinary Internal Medicine.

[12] Siriphanporn Y, Wiratsudakul A, Kasemsuwan S & et al. 2025. Retrospective evaluation of the prognostic utility of clinical and laboratory findings in hospitalized cats with pancreatitis. Animals, 15(7): 1060.

[13] Quimby JM. 2016. Update on medical management of clinical manifestations of chronic kidney disease. Veterinary Clinics: Small Animal Practice.

[14] Willard MD & Twedt DC. 2011. Small animal clinical diagnosis by laboratory methods. National Institutes of Health.

[15] Carvalho L, Kelley D, Labato MA & et al. 2021. Hyperammonemia in azotemic cats. Journal of Feline Medicine and Surgery.

[16] Dulude MD, Ford SL & Lynch H. 2024. Association between pancreatitis and chronic kidney disease in cats: A retrospective review. Journal of the American Veterinary Medical Association, 262(5): 615.

[17] Tolbert K & Stubbs E. 2024. Rational use of gastroprotectants in cats: An evidence-based approach. Journal of Feline Medicine and Surgery.

[18] De Santis F, Boari A, Dondi F & Crisi PE. 2022. Drug-dosing adjustment in cats with chronic kidney disease. Animals, 12(3): 262.

[19] Norsworthy GD, Estep JS & Hollinger C. 2015. Prevalence and underlying causes of histologic abnormalities in cats suspected to have chronic small bowel disease. Journal of the American Veterinary Medical Association, 247(6): 629–636..