

# Pre-renal chronic kidney disease in a domestic cat presenting with FLUTD-like symptoms<sup>†</sup>

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**ABSTRACT:** Chronic kidney disease (CKD) is a progressive condition in aging cats that often presents with signs of overlapping feline lower urinary tract disease (FLUTD). Early recognition and differentiation are crucial for its management and prognosis. This case study discusses a 5-year-old cat with CKD who initially showed FLUTD-like symptoms, including three days of anuria, dehydration, and anorexia. Diagnostics, including examination, ultrasonography, urinalysis, hematology, and serum biochemistry, revealed elevated creatinine, amylase, and BUN levels, indicating pre-renal impairment and decreased GFR. Treatment involved cystocentesis, hepacartine, azody, ippakitine, vitamin B-complex, and a renal-support diet (Royal Canin Renal Wet Food). After two weeks, the cat showed clinical improvement with clear urine and normalized biochemistry.

## Keywords:

chronic kidney disease, cystocentesis, renal diet, prerenal, feline lower urinary tract disease

## ■ INTRODUCTION

Chronic Kidney Disease (CKD) is a progressive disorder marked by reduced kidney function to excrete waste, concentrate urine, and maintain electrolyte balance over three months (Reynolds *et al.*, 2013). In cats, CKD prevalence is 1–3% and increases with age (IRIS, 2019). This disease involves irreversible renal damage caused by azotemia. According to the International Renal Interest Society (IRIS), CKD ranges from non-azotemic (stage 1) to end-stage renal disease (stage 4) with severe azotemia (Reynolds *et al.*, 2013).

Renal disease diagnosis is based on the detection of azotemia from pre-renal, renal, or post-renal causes. Prerenal azotemia occurs with decreased renal perfusion, as confirmed by urine-specific gravity. Structural disorders, such as polycystic kidney disease, may cause similar findings (Polzin, 2013). In renal azotemia, impaired urine concentration results in isosthenuria (specific gravity 1.008–1.013), indicating renal function loss. Postrenal azotemia occurs due to urinary tract obstruction, which impairs waste excretion (Fischer *et al.*, 2009).

Early diagnosis of feline CKD remains challenging owing to subtle signs and biochemical limitations in detecting renal dysfunction. Identifying diagnostic indicators that combine hematological, biochemical, and urinalysis profiles may improve disease staging. This study describes biochemical changes in feline CKD, focusing on azotemia patterns and urine-specific gravity. This case report provides insights into CKD diagnosis and laboratory data integration for management.

## ■ CASE

**Anamnesis and Signalment:** A domestic cat presented to the Veterinary Teaching Hospital, Universitas Padjadjaran. Previously diagnosed with FLUTD at a private clinic, the cat showed anuria for three days, dehydration, and loss of appetite. **Physical Examination:** The cat weighed 3.5 kg and had a temperature of 37.9°C. The skin and coat appeared sticky and dull, indicating poor grooming. Dehydration was confirmed by delayed CRT and skin turgor of >2 s. The oral mucous membranes were pale pink, suggesting anemia. **Clinical Workup:** Diagnostic examinations included ultrasonography (Figure S1), hematology, biochemical (Table S2), and urinalysis (Table S3) to assess renal function and identify the cause of azotemia. **Diagnosis:** CKD secondary to pre-renal injury from dehydration and urinary retention due to FLUTD. **Differential Diagnosis:** Acute kidney injury (AKI), obstructive uropathy from FLUTD, pyelonephritis, and post-renal azotemia. AKI was unlikely given the chronic renal findings. Obstructive uropathy and postrenal azotemia were excluded by ultrasound, which showed no obstruction. Pyelonephritis was ruled out due to the absence of fever. **Prognosis:** The prognosis was guarded to fair based on renal damage. With dietary management and hydration, stabilization was achievable, although complete recovery was unlikely due to CKD. **Treatment and Management:** The protocol included cystocentesis for urinary retention, antibiotics, hepacartine,

Received: 19-05-2025 | Revised: 23-06-2025 | Accepted: 28-06-2025

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azodyl, ippakitine, and vitamin B-complex. A renal-support diet (Royal Canin Renal Wet Food) managed nutrition. Treatment aims to restore hydration, reduce azotemia, and support hepatic and renal functions.

## ■ RESULTS AND DISCUSSION

Hematological examination (Table S1) revealed leukocytosis, neutrophilia, monocytosis, and anemia. Leukocytosis and neutrophilia indicate infection, tissue necrosis, and neoplasia, respectively (Lucroy, 1999). Monocytosis reflects chronic inflammation or infection (Marionneaux 2020). Decreased RBC count and hematocrit in cats with CKD indicate non-regenerative anemia from reduced erythropoietin production (Chalhoub *et al.*, 2011). These findings suggest chronic inflammation, which is consistent with a systemic disease.

Serum biochemistry showed elevated BUN and creatinine, indicating pre-renal azotemia from decreased renal perfusion. In CKD, reduced glomerular filtration decreases protein metabolite excretion. BUN-to-creatinine elevation indicates dehydration or muscle wasting (Polzin, 2011). Elevated BUN and creatinine with polyuria suggest chronic renal dysfunction. A renal-support diet was prescribed for CKD stages 3-4. Monitor response through weight, BCS, and serum albumin (Polzin, 2011). Hepacartine supports cardiovascular health through l-carnitine (Savic *et al.*, 2020). Azodyl reduces uremic toxins, while ippakitine binds intestinal phosphate. Post-treatment tests showed decreased BUN, amylase, creatinine, and bilirubin levels, though above reference range, indicating partial renal recovery.

Urinalysis revealed ketonuria, elevated creatinine levels, microalbuminuria, proteinuria, nitrituria, leukocyturia, glucosuria, and alkaline pH, indicating impaired glomerular filtration (Van Vetloo, 2025). Proteinuria is a marker for poor feline CKD outcomes and damages the renal tubules. Tests to determine renal impairment. Urinary retention may lead to renal failure (Lew-Kojrys *et al.*, 2017). During catheterization, an intravesical mass blocked the urethra and required cystocentesis. Cystocentesis showed cloudy urine with purulent material (Figure 1). The urine cleared in subsequent days. Small blood clots appeared on day 9. By day 13, the urine output normalized without blood clots.

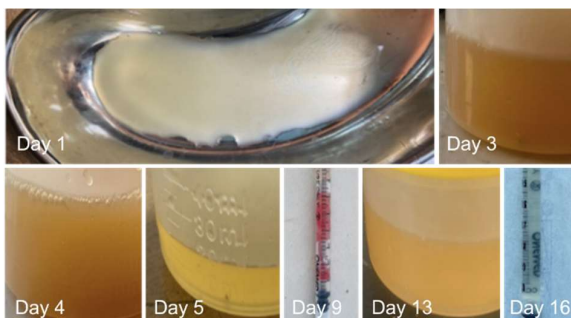


Figure 1. The results of cystocentesis on day 1 and the progression of urination over a two-week treatment period showed gradual improvement in urine clarity following therapeutic management.

## ■ CONCLUSION

A domestic cat presented to the Veterinary Teaching Hospital, Padjadjaran University, with three days of anuria, dehydration, and loss of appetite. Clinical findings indicate that pre-renal impairment is likely associated with pancreatic dysfunction. Symptomatic and causative treatments lead to marked clinical improvement, suggesting a favorable prognosis.

## ■ ASSOCIATED CONTENT

### Supporting Information

†The sonogram, hematology, blood biochemistry, and urinalysis were submitted in PDF form as supporting information.

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