ACTA VETERINARIA INDONESIANA
P-ISSN 2337-3202, E-ISSN 2337-4373

Vol. 13, No. 3: 191-198, November 2025

Research

The Examination of BUN and Creatinine Value in Cats with Feline Lower Urinary Tract Disease

Palestin Palestin[1,2]*, Intan Permatasari Hermawan[1], Retina Yunani[1]

[1]Faculty of Veterinary Medicine, Universitas Wijaya Kusuma Surabaya, Surabaya, 60225, Indonesia
[2]K and P Clinic, Surabaya, Indonesia
*Corresponding author: drh.palestin@uwks.ac.id
Received: 9 February 2024, Accepted 29 September 2025

ABSTRACT

Feline Lower Urinary Tract Disease (FLUTD) is one of the most common diseases affecting cats. One type of FLUTD that often occurs is urinary tract obstruction. Urinary tract obstruction makes it difficult for cats to urinate, causing urea and creatinine to be reabsorbed into the bloodstream. This study aimed to examine of Blood Urea Nitrogen (BUN) and creatinine values in cats with Feline Urinary Tract Disease (FLUTD). Cats with FLUTD often had kidney problems such as azotemia and chronic kidney disorders called Chronic Kidney Disease (CKD). Both conditions can be seen from high levels of BUN and Creatinine in the blood. The BUN and Creatinine levels were examined by taking blood serum from cats with FLUTD using a 3 cc disposable syringe and accommodated in a plain tube and then centrifuged at 3000 rpm for 10 minutes. The tool used to perform BUN and creatinine examinations is the VetTest Idexx Laboratory® blood chemistry examination tool. The result of the examination on 25 cats was 64% (16/25) had an increase in BUN values and 36% (9/25) had an increase in creatinine values. BUN and creatinine are waste products of metabolism that are filtered by the glomerulus and excreted in the urine. Elevated levels of BUN and creatinine in cats with FLUTD are due to the cat's inability to urinate as a result of urinary tract obstruction or decreased glomerular filtration rate. Based on the results, it can be concluded that cats with FLUTD will also have kidney problems, and additional therapy is needed to reduce BUN and creatinine levels.

Keywords: FLUTD, BUN, Creatinine, Azotemia

ABSTRAK

Penelitian ini bertujuan untuk mengetahui nilai Blood Urea Nitrogen (BUN) dan kreatinin pada kucing penderita Feline Urinary Tract Disease (FLUTD). Kucing dengan FLUTD seringkali mengalami gangguan ginjal seperti azotemia dan kelainan ginjal kronis yang disebut Chronic Kidney Disease (CKD) . Kedua kondisi tersebut terlihat dari tingginya kadar BUN dan Kreatinin dalam darah. Kadar BUN dan Kreatinin diperiksa dengan mengambil serum darah kucing penderita FLUTD menggunakan spuit sekali pakai berukuran 3 cc dan ditampung dalam tabung kemudian disentrifugasi dengan kecepatan 3000 rpm selama 10 menit. Alat yang digunakan untuk melakukan pemeriksaan BUN dan kreatinin adalah alat pemeriksaan kimia darah VetTest Idexx Laboratory®. Hasil pemeriksaan pada 25 ekor kucing terdapat 64% (16/25) mengalami peningkatan nilai BUN dan 36% (25/9) mengalami peningkatan nilai kreatinin. Berdasarkan hasil penelitian, dapat disimpulkan bahwa kucing dengan FLUTD juga akan mengalami gangguan ginjal, sehingga diperlukan terapi tambahan untuk menurunkan kadar BUN dan kreatinin.

Kata kunci: FLUTD, BUN, Kreatinin, Azotemia

INTRODUCTION

Feline Lower Urinary Tract Disease (FLUTD) is a disease that affects the urethra and bladder of cats (Nururrozi et al., 2020; He et al., 2022). Symptoms of FLUTD include dysuria, hematuria, stranguria, pollakiuria, and (Piyarungsri et al., 2020; Kartashov et al., 2021). Cats with Feline Lower Urinary Tract Disease (FLUTD) can be caused by various factors, including feline idiopathic cystitis, interstitial cystitis, urolithiasis, bacterial infections of the urinary tract, anatomical malformations of the urinary system, neoplasia, behavioral disorders, and neurological disorders such as dysnergia reflex (Azhar et al., 2022; Kovarikova et al., 2020; Byregowda et al., 2023; Abdel-saeed et al., 2020). FLUTD can also be caused by urethral obstruction due to mucus and stone blockages (Lew-Kojrys et al., 2018; Reines & Wagner, 2018) (Jackson et al., 2023).

The prevalence of FLUTD is estimated to range from 1.5% to 2.2% of the domestic cat population. Feline idiopathic cystitis (FIC) is the most common condition, responsible for 54-69% of FLUTD cases, and is diagnosed after excluding other causes of FLUTD (Jackson et al., 2023). FLUTD is reported to occur in 0.34–0.64% of the cat population, and 4–10% of the reasons owners bring their cats to the veterinarian are also reported (Novitasari et al., 2023).

FIC is a poorly understood yet complex condition, often linked to management practices and environmental stressors, such as sudden environmental changes, the introduction of new pets or people to the home, and owner stress. Neuroendocrine abnormalities combined with stressors are believed to be a primary cause in the development of FIC (Jackson et al., 2023).

In addition to the various clinical symptoms above, cats with FLUTD also experience azotemia (One et al., 2023). Azotemia is an increase in the concentration of nitrogen-containing substances in the blood, especially blood urea nitrogen (BUN) and creatinine.

Azotemia can be classified into three types : prerenal, renal, and postrenal. Prerenal azotemia occurs when decreased renal perfusion causes a decrease in glomerular filtration rate. Renal azotemia can occur when nephron damage is directly caused by renal parenchymal disease. Postrenal azotemia can develop due to obstruction in the lower urinary tract, causing accumulation, retention, and impaired excretion of urine from the body (Pridayanti et al., 2023; Gülersoy & Ekici, 2020).

Blood Urea Nitrogen (BUN) is a component that describes the number of nitrogen atoms in the blood combined with urea (Arifianto et al., 2020). Urea is synthesized in the liver, originating from ammonia, which is produced by amino acid deamination. Urea is a small molecule that easily diffuses throughout the body's fluid compartments, is freely filtered in the glomerulus but is then mostly reabsorbed in the renal tubules (Arifianto et al., 2020; Syme, 2016).

Creatinine is a small molecule produced from the breakdown of phosphocreatine, also known as creatine phosphate (Syme, 2016). Arifianto et al. (2020) explain that creatinine is a byproduct of muscle metabolism that is freely filtered by the glomerulus and not reabsorbed by the renal tubules.

The levels of urea and creatinine can be affected by factors unrelated to kidney function, including dehydration and consumption of a high-protein diet. Since urea is reabsorbed passively in the renal tubules, its concentration in the plasma may rise when the tubular flow rate is reduced, as seen in cases of dehydration or hypovolemia (Finch, 2014).

This study aims to determine the levels of BUN and creatinine in cats with FLUTD. This examination is crucial as it is related to the further therapy that will be administered to cats with FLUTD, thereby improving the prognosis of the disease. Additionally, veterinarians and owners can determine early on whether there are any kidney issues.

MATERIALS AND METHODS

Sample

The animals used in this study were 25 adult cats suffering from FLUTD (with symptoms of urethral obstruction), consisting of 3 female cats and 22 male cats.

Diagnostic Technique

The diagnosis of FLUTD in this study was confirmed by observing the owner's medical history and clinical symptoms, supported by other supporting examinations such as microscopic observation of urine to determine whether there were sediment in the urine (blood, plaque, or calculi). All samples were obtained from patients who presented with complaints of difficulty urinating.

Unblocking Urethra

Close-ended catheter measuring 1.0 mm x 145 mm, lubricant, RL infusion for flushing, olive tip

catheter, 3cc and 10cc syringes, intravenous catheter, infusion set.

Blood Sampling

Blood samples were collected using the vena puncture technique through the cephalic vein (Figure 1) using a 3 cc syringe. The blood obtained was then placed in a plain tube (Figure 2), after which the tube containing the blood was placed in a Corona® centrifuge at a speed of 3000 rpm for 10 minutes to obtain blood serum (Figure 3).

BUN and Creatinine Level Testing

BUN and creatinine levels were tested using the VetTest Chemistry Analyzer by IDEXX Laboratories® (Figures 4 and 5).

Therapy for Cats with FLUTD

Tramadol as an analgesic at a dose of 3 mg/kg BW administered intravenously for 5 days, 3 times a day, fluid therapy using crystalloid fluids, urinary tract supplements, kidney supplements, and special food for urinary problems.

Figure 1. Blood collection from the cephalic vein (green arrow) using a 3 cc syringe

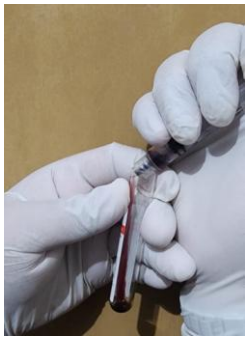

Figure 2. The blood obtained is placed in a plain tube.

Figure 3. Blood is centrifuged using a Corona® centrifuge at a speed of 3000 rpm for 10 minutes to obtain the serum.

Figure 4. BUN and Creatinine Level Testing using the VetTest Chemistry Analyzer by IDEXX Laboratories®

Figure 5. BUN and Creatinine Reagents

Data

The research data were not analyzed but presented in tabular form.

RESULTS

The results of BUN and creatinine tests in cats suffering from FLUTD are presented in Table 1. Based on the results of BUN and creatinine tests in 25 cats suffering from FLUTD, 16 cats experienced an increase in BUN levels, 1 cat experienced a decrease in BUN levels, and 9 cats experienced an increase in creatinine levels, which means that 9 cats experienced azotemia.

DISCUSSION

Based on the results of the study, cats with FLUTD also experienced an increase in BUN and creatinine levels. From 25 cats with FLUTD, 22 were male and 3 were female. All of these cats experienced urethral of the 25 blood serum samples from cats with FLUTD (with symptoms of urethral obstruction) that were examined, 36% or 9 of them experienced postrenal azotemia. 64% or 16 cats experienced an increase in BUN values.

As shown in Table 1, the highest BUN and creatinine values were found in Tam Tam (BUN: 130 mg/dl, creatinine: 12.6 mg/dl) and Cece (BUN: 130 mg/dl, creatinine: 11.0 mg/dl). This occurred because their urination difficulties were not treated immediately. Based on complaints from their owners, they experienced difficulty urinating for more than 5 days. This condition was different from that experienced by Bubu (BUN: 18 mg/dl, Creatinine: 1.1 mg/dl) and Justin (BUN: 18 mg/dl, Creatinine: 1.5 mg/dl). Their owners immediately recognized that they were experiencing difficulty urinating, so the condition was promptly and effectively addressed.

Different conditions occurred in Briso (BUN: 15 mg/dl, Creatinine: 0 mg/dl) and Bomi (BUN: 29 mg/dl, Creatinine: 0.7 mg/dl), both of whom experienced a decrease in creatinine levels. This could be due to a decrease in muscle mass or a low-protein diet.

Table 1. Kadar BUN and Creatinine

Name of Cat	Sex	BUN (mg/dL)	Normal Value (mg/dL)	Creatinine (mg/dL)	Normal Value (mg/dL)
Bomi	Male	29		0,7 (L)	
Bembi	Male	34		0.8	
Bubu	Male	18		1.1	
Stark	Male	20		1.2	
Steve	Male	36 (H)		2.3	
Cleo	Male	51 (H)		3,9 (H)	
Simba	Male	130 (H)		-	
Tam Tam	Male	130(H**)		12,6 (H**)	
Cece	Female	130(H**)		11,0 (H**)	
Chiro	Male	33		1.5	
Miko	Male	109 (H**)		6 (H**)	
Chill	Male	32		1.8	
Hima	Female	62 (H**)	16.00 – 36.00	2	0.80 – 2.40
Nggora	Male	47 (H)		1.8	
Bakpao	Male	84 (H**)		4 (H)	
Briso	Male	15 (L)		0 (L)	
Messi	Male	92 (H**)		3,5 (H)	
Piko	Male	52 (H*)		5,1 (H)	
Otong	Male	94 (H**)		7,0 (H**)	
Moly	Male	128 (H**)		1.5	
Whity	Male	67 (H**)		7,4 (H**)	
Justin	Male	18		1.5	
Artemis	Female	27		1.9	
Steve	Male	36 (H)		2.3	
Snow	Male	46 (H)		2	

Reference by VetTest Chemistry Analyzer by IDEXX Laboratories®

Additionally, this condition could also be caused by a decrease in creatinine production due to reduced kidney function (Wati et al., 2024).

Urethral obstruction (UO) is a frequent complication of Feline Lower Urinary Tract Disease (FLUTD), caused either by physical blockages like struvite crystals or mucus plugs, or by functional blockages resulting from urethral spasms associated with inflammation. Male cats are more commonly affected due to their longer and narrower urethra (Jackson et al., 2023).

Male cats are more commonly affected due to their urethral structure and typical behavior, which can lead to urinary obstruction (UO). Urethral plugs account for as many as 60% of UO cases in cats; other possible causes include urinary stones, idiopathic UO, urethral strictures from injury, tumors, and foreign objects blocking the urethra (Jerabkova et al., 2024).

Obstruction of the urethra can cause hemodynamic changes. Hemodynamic alterations in urinary tract obstruction (UTO) are influenced by both the site and the extent of the obstruction. Initially, the rise in intratubular pressure is counterbalanced by an increase in renal blood flow, mediated by the renal synthesis and release of intrinsic prostaglandin E2 (PGE2), which serves to preserve an adequate glomerular filtration rate (GFR) (Pérez-Aizpurua et al., 2024).

This compensatory mechanism, however, is transient and typically persists for no longer than one to two hours, after which renal blood flow progressively declines while intratubular pressure continues to rise. After approximately three to four hours, a marked reduction in renal perfusion becomes evident as a consequence of sustained elevation in intratubular pressure (Pérez-Aizpurua et al., 2024).

The subsequent decrease in renal vascular supply results in a concomitant decline in intratubular pressure. Impaired renal blood flow further contributes to a reduction in GFR and a redistribution of intrarenal perfusion from the cortical toward the medullary region (Pérez-Aizpurua et al., 2024).

Ureteral obstruction may occur at the junction between the renal pelvis and the ureter, known as the ureteropelvic junction (proximal ureter), or at the point where the ureter connects to the urinary bladder, called the ureterovesical junction (Arifianto et al., 2020).

Ureteral obstruction may occur at the junction between the renal pelvis and the ureter, known as the ureteropelvic junction (proximal ureter), or at the point where the ureter connects to the urinary bladder, called the ureterovesical junction (Arifianto et al., 2020).

These obstructions can be caused by congenital conditions such as lumen narrowing (stenosis), the presence of kidney stones (calculi), or twisting (torsion) due to trauma. Early diagnosis is essential, as prolonged blockage can decrease the effectiveness of treatment and make it more difficult for the kidney to regain normal function (Arifianto et al., 2020).

Obstructions may be located either in the proximal region (ureteropelvic junction) or the distal region (ureterovesical junction). Each location affects the kidneys differently and requires a specific therapeutic approach, making it crucial to determine the exact site of obstruction prior to surgery (Arifianto et al., 2020). This condition can lead to azotemia (increase value of BUN dan Creatinine).

Severe metabolic imbalances, such as hyperkalemia, metabolic acidosis, and hypocalcemia, can develop. Hyperkalemia is the most common life-threatening complication and may result in bradycardia and cardiac arrhythmias, exacerbated by hypocalcemia. Uremia typically occurs within 24–48 hours when urine output is complete and acute. Ongoing gastrointestinal losses and decreased fluid intake (due to vomiting and anorexia) can lead to significant dehydration and hypovolemia. If not treated promptly, complete urine output can cause severe bradycardia, bladder rupture, urinoma, concurrent shock, and death (Taylor et al., 2025).

Routine fluid administration is a standard component in the treatment of feline UO to restore circulation and normalize electrolyte and acid-base status. The conventional wisdom held that 0.9% saline was superior for treating *hyperkalemia* compared to balanced replacement fluids (like LRS or Normosol-R) due to its lack of potassium. Yet, this belief was challenged when the acidifying properties of PSS were recognized, prompting worries that it might worsen the metabolic acidosis typically seen in obstructed cats instead of improving it (Wobeser, 2020).

Azotemia can occur when there is an obstruction to urine collection, retention, or excretion caused by any factor beyond the renal tubules. If waste products are not properly eliminated through urine, it can lead to serious disturbances in fluid balance, electrolytes, and acid-base homeostasis that may become life-threatening (Gülersoy & Ekici, 2020).

Azotemia is classified into three conditions, namely prerenal azotemia, renal azotemia, and

postrenal azotemia. Prerenal azotemia can be caused by increased protein catabolism and decreased renal perfusion, which can increase urine specific gravity. Renal azotemia occurs when 75% of nephrons lose function and is characterized by a large increase in BUN and creatinine. Postrenal azotemia is characterized by the onset of oliguria or anuria caused by urethral obstruction or rupture (Mardasella, 2021).

The conditions such as those shown in Table 1, an increase in BUN and creatinine can cause postrenal azotemia. Postrenal azotemia can be caused by an infection in the lower urinary tract, which can inhibit the normal collection and excretion of urine from the body (Pridayanti et al., 2023). This increase occurs due to the accumulation of toxic chemicals in the bloodstream that is blocked in the urethra, which should be physiologically excreted (Apritya et al., 2017). Azotemia can affect the glomerular filtration rate (Purnamaningsih et al., 2024).

The glomerular filtration rate (GFR) plays a crucial role in generating the ultrafiltrate, which is then subjected to selective reabsorption and secretion within the renal tubules via tubular transport mechanisms (Finch, 2014).

The assessment of glomerular filtration rate (GFR) is considered the most accurate indicator of the kidney's functional mass. Single nephron GFR (snGFR) is calculated by multiplying the effective ultrafiltration pressure with the ultrafiltration coefficient. The effective ultrafiltration pressure is influenced by both hydraulic and oncotic pressures within the glomerular capillaries and Bowman's capsule (Finch, 2014).

Consequently, significant hypotension can lead to a reduction in GFR due to decreased hydraulic pressure in the glomerular capillaries, which cannot be fully offset by the kidney's autoregulatory mechanisms. Additionally, an obstruction in the lower urinary tract raises hydraulic pressure in Bowman's space, thereby lowering GFR. The ultrafiltration coefficient itself is dependent on the permeability of the glomerular membrane and the surface area of the capillaries (Finch, 2014).

The association between creatinine levels and GFR follows a curvilinear pattern, specifically a rectangular hyperbola. At the extremes of this curve, significant shifts in one variable lead to minimal changes in the other. Therefore, during the initial phases of kidney disease, considerable declines in GFR may not noticeably affect plasma creatinine levels, which can still fall within the normal range. Conversely, in the later stages of renal failure, even a slight decrease in GFR can result in a marked rise in creatinine levels (Kovarikova, 2018).

In this study, the actions taken to treat FLUTD cases were catheterization, fluid therapy, and administration of medication. Catheterization is one of the procedures that can be performed in the event of urethral obstruction, allowing urine to be drained immediately. Catheterization is carried out by retracting the penis from the prepuce and aligning it parallel to the spine to minimize the risk of injury to the urinary tract. Prior to insertion, the catheter should be thoroughly cleaned with an antiseptic and lubricated with a liquid lubricant (Azhar et al., 2022).

The catheter is inserted with the assistance of a flushing technique using a syringe filled with saline solution, which is slowly introduced to help expand the urethra and facilitate easier insertion. Additionally, flushing serves to clear cellular debris from the urethra, particularly in cases involving blockage (Azhar et al., 2022). Once the catheter is in place, the next step is to perform a microscopic examination to determine the exact cause of the urethral obstruction.

In addition to catheterization, intravenous fluids must be administered immediately to help lower BUN and creatinine levels in the blood and be excreted through urine.

Intravenous (IV) fluid administration is essential for treating dehydration and hypovolemia, improving renal perfusion, and managing hyperkalemia. Fluid therapy should be initiated promptly and not postponed until a urinary catheter is inserted, as early intervention supports renal perfusion and lowers serum potassium levels. Suitable crystalloid fluids include 0.9% saline or balanced isotonic solutions that contain 4–5 mmol/L of potassium (Taylor et al., 2025).

There is evidence indicating that balanced isotonic crystalloids might be more effective in correcting acidosis quickly; however, the type of fluid used (either 0.9% saline or balanced isotonic solutions) does not appear to affect the normalization of serum potassium levels (Taylor et al., 2025).

The therapy used in this study was the administration of Tramadol analgesic at a dose of 3 mg/kg BW IV 3 times a day for 5 days, urinary tract supplements, namely N-acetyl D-glucosamine, and kidney supplements in the form of various amino acids, including Glycine, L -Aspartic acid, L-Glutamic acid, L-Glutamine, L-Carnosine, L-Histidine, L-Arginine.

Increased BUN and creatinine levels can cause azotemia. In cases of Feline Urinary Tract Disease (FLUTD), cats may experience postrenal azotemia. Postrenal azotemia often occurs as a result of disease in the lower urinary tract, especially with symptoms of urethral obstruction.

"The author declares that there is no conflict of interest with the parties involved in this research."

REFERENCES

Abdel-saeed, H., Tahon, R., & Farag, H. S. (2020). *Diagnostic and epidemiological studies on obstructive feline lower urinary tract disease (FLUTD) with special reference to anatomical findings in Egyptian tomcats DIAGNOSTIC AND EPIDEMIOLOGICAL STUDIES ON OBSTRUCTIVE FELINE LOWER URINARY TRACT DISEASE (*. *April*. https://doi.org/10.15547/bjvm.2019-0096.

Arifianto, D., Adji, D., Sutrisno, B., & Rickiawan, N. (2020). Renal Histopathology , Blood Urea Nitrogen and Creatinine Levels of Rats With Unilateral Ureteral Obstruction. *Indonesian Journal of Veterinary Sciences*, *1*(1), 1–9. https://doi.org/10.22146/ijvs.v1i1.46515

Apritya, D., Yunani, R., Widyawati, R., Hewan, L. K., Patologi, L., Hewan, K., Lower, F., & Tract, U. (2017). Analisis Urin Kasus Urolithiasis pada kucing tahun 2017. *Agreteriner*, *6*(1), 82–85.

Azhar, A. P. N., Wardhani, L. D. K., & Palestin, P. (2022). Journal of Applied Veterinary Science and Tecnology. *Journal of Applied … , 03*, 18–21. https://doi.org/10.20473/javest.V3.01.2022.18-21

Byregowda, N., Veterinary, K., Veterinary, K., Habeeb, B. P., Veterinary, K., Gopalan, U. C., & Veterinary, K. (2023). *Evaluation of etiology , risk factors and clinical signs of feline lower urinary tract disease (FLUTD) in Northern Kerala #*. *March*. https://doi.org/10.51966/jvas.2023.54.1.269-274

Finch, N. (2014). Measurement of glomerular filtration rate in cats: Methods and advantages over routine markers of renal function. *Journal of Feline Medicine and Surgery*, *16*(9), 736–748. https://doi.org/10.1177/1098612X14545274.

Gülersoy, E., & Ekici, Y. E. (2020). Acute Postrenal Azotemia In a Cat. *Alexandria Journal of Veterinary Sciences*, *64*(1), 1–4. https://doi.org/10.5455/ajvs.77021.

He, C., Fan, K., Hao, Z., Tang, N., Li, G., & Wang, S. (2022). *Prevalence , Risk Factors , Pathophysiology , Potential Biomarkers and Management of Feline Idiopathic Cystitis : An Update Review*. *9*(June). https://doi.org/10.3389/fvets.2022.900847.

Jackson, K. A., Collins, K. E., Kim, T. Y., & Donaldson, R. E. (2023). Incidence of feline idiopathic cystitis and urethral obstruction during COVID-19 human movement restrictions in Queensland, Australia. *Journal of Feline Medicine and Surgery*, *25*(12). https://doi.org/10.1177/1098612X231214931.

Jerabkova, P., Mattoon, J. S., Darveshi, B. R. M., & Owen, T. J. (2024). Recurrent lower urinary tract signs in a 4-year-old male castrated domestic longhair cat. *Journal of the American Veterinary Medical Association*, *262*(9), 1265–1268. https://doi.org/10.2460/javma.24.03.0173.

Kartashov, S., Bekker, O., Oboeva, M., &*syndrome in cats changes in urological*. *02027*.Kartashova, E. (2021). *Morphofunctional.*

Kovarikova, S., Brno, P. S., & Simerdova, V. (2020). *Clinicopathological characteristics of cats with signs of feline lower urinary tract disease in the Czech Republic*. *March*. https://doi.org/10.17221/146/2019-VETMED.

Kovarikova, S. (2018). Indirect markers of glomerular filtration rate in dogs and cats: A review. *Veterinarni Medicina*, *63*(9), 395–412. https://doi.org/10.17221/77/2017-VETMED

Lew-Kojrys, S., Mikulska-Skupien, E., Snarska, A., Krystkiewicz, W., & A, P. (2018). *Evaluation of clinical signs and causes of lower urinary tract disease in Polish cats*. *July 2017*. https://doi.org/10.17221/170/2016-VETMED.

Mardasella, A. (2021). Gagal ginjal kronis pada Kucing Domestik Rambut Pendek. *Media Kedokteran Hewan*, *32*(1), 29. https://doi.org/10.20473/mkh.v32i1.2021.29-39.

Novitasari, S. P., Yunita, M. N., Hamid, I. S., & Wibawati, P. A. (2023). *Faktor Risiko dan Insidensi Feline Lower Urinary Tract Disease (FLUTD) di Klinik Sahabat Satwa Genteng*. *6*(1), 15–20. https://doi.org/10.20473/jmv.vol6.iss1.2023.15-20.

Nururrozi, A., Yanuartono, Y., Sivananthan, P., & Indarjulianto, S. (2020). Evaluation of lower urinary tract disease in the Yogyakarta cat population , Indonesia. *Veterinary World*, *13*, 1182–1186.

One, T., Concept, H., Mustafa, B., Shehta, A., Gouda, H. ,&Shety, T. (2023). *MONITORING THE INFLAMMATORY PROCESS OF FELINE*. *60*(Suppl 25), 173–183. https://doi.org/10.26873/SVR-1577-2022.

Piyarungsri, K., Tangtrongsup, S., Thitaram, N., Lekklar, P., & Kittinuntasilp, A. (2020). Prevalence and risk factors of feline lower urinary tract disease in. *Scientific Reports*, 1–9. https://doi.org/10.1038/s41598-019-56968-w.

Pridayanti, N. K. N., Anthara, M. S., & Widyastuti, S. K. (2023). Laporan Kasus : Infeksi Saluran Kemih Bawah Penyebab Azotemia Post- Renal pada Kucing Persia Campuran. *Buletin Veteriner Udayana*, *15*(4), 647–655. https://doi.org/10.24843/bulvet.2023.v15.i04.p17.

Reines, B. P., & Wagner, R. A. (2018). Resurrecting FUS : Adrenal Androgens as an Ultimate Cause of Obstruction in Neutered Cats. *Frontiers in Veterinary Science*, *5*(September), 1–7. https://doi.org/10.3389/fvets.2018.00207.

Syme, H. M. (2016). BSAVA Manual of Canine and Feline Clinical Pathology. In E. Villiers & J. Ristic (Eds.), *British Small Animal Veterinary Association* (Third Edit, pp. 219–221). British Small Animal Veterinary Association Woodrow House, 1 Telford Way, Waterwells Business Park, Quedgeley, Gloucester GL2 2AB A.

Taylor, S., Boysen, S., Buffington, T., Chalhoub, S., Defauw, P., Delgado, M. M., Gunn-Moore, D., & Korman, R. (2025). 2025 iCatCare consensus guidelines on the diagnosis and management of lower urinary tract diseases in cats. *Journal of Feline Medicine and Surgery*, *27*(2), 1–36. https://doi.org/10.1177/1098612X241309176.

Wati, D. N., Farma, S. A., & Cahyanti, N. (2024). Analisis fungsi ginjal kasus Feline Lower Urinary Tract Disease (FLUTD) pada kucing di UPTD rumah sakit hewan Sumatera Barat. *Prosiding Seminar Nasional Biologi*, *4*(1), 119–124. https://semnas.biologi.fmipa.unp.ac.id/index.php/prosiding/article/view/916.

Wobeser, G. (2020). In-hospital medical management of feline urethral obstruction: A re view of recent clinical research. *Canadian Veterinary Journal*, *50*(11), 1169–1176.