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Urinary Tract Emergencies in Small Animals – A Review

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Abstract
Urinary tract emergencies in dogs and cats, including urinary tract obstruction, uroperitoneum, acute kidney injury (AKI), and feline urethral obstruction (FUO), represent critical conditions requiring rapid diagnosis and intervention. Urethral and ureteral obstructions commonly result from urolithiasis in dogs and urethral plugs in cats, leading to metabolic derangements such as hyperkalemia, metabolic acidosis, and azotemia. Uroperitoneum arises from lower urinary tract rupture, confirmed by diagnostic imaging and fluid biochemical ratios. AKI develops secondary to ischemia or nephrotoxicity, exhibiting elevated renal biomarkers and azotemia. Feline urethral obstruction, often related to stress-induced FLUTD, necessitates stabilization, catheterization, pain control, and environmental management. Prompt recognition and correction of electrolyte imbalances, fluid therapy, and appropriate surgical or medical interventions are vital to prevent life-threatening sequelae and ensure successful recovery.

Keywords: Urethral obstruction, uroperitoneum, acute kidney injury

Introduction
The most common and important urinary tract emergencies occurring in dogs and cats are

- Urinary Tract Obstruction
- Uroperitoneum
- Acute Kidney Injury
- Feline Urethral Obstruction

Urinary Tract Obstruction
Urinary tract obstruction in dogs and cats is a life-threatening emergency and are mostly of two types: Ureteral and Urethral. The most common cause of urinary tract obstruction in cats are mucous plugs made up of struvites, whereas in dogs, the most common cause is urolithiasis. Neoplasia, such as squamous cell carcinoma and transitional cell carcinoma are also other important causes.
The most common changes in urinary tract obstruction are

- Metabolic acidosis – It occurs due to the inability to excrete Hydrogen ions in urine.
- Hyperkalemia – It occurs due to the inability to excrete potassium ions in urine, thereby exhibiting cardiovascular effects such as bradycardia, tall T wave, shortened QT interval, ST depression and inability of the heart to repolarize after depolarization.
- Hypocalcemia – Metabolic acidosis leads to the shift of calcium from the protein bound fraction to the ionized fraction, thereby leading to neuromuscular hyperexcitability, less cardiac contractility and peripheral vasodilation.
- Hyperphosphatemia – It occurs due to less renal clearance of phosphorus, thereby exaggerating the effects of metabolic acidosis and hypocalcemia.

The most common signs associated with Urinary Tract Obstruction in dogs and cats include stranguria, lethargy, anorexia, extremely firm bladder and bradycardia.
Diagnosis can be done by diagnostic imaging technologies like radiography, contrast

radiography, antegrade pyelography, abdominal ultrasonography, computed tomography for visualization of obstruction site and distention of the ureter/urethra anterior to the obstruction. Packed Cell Volume can be elevated due to dehydration from anorexia. Blood Urea In Butt view of radiography (especially in males), urethral calculi can be visualized. Nitrogen can be elevated due to post renal azotemia. Urinalysis should be carried out to assess the type of crystals present in the urine.

Treatment should be carried out in first providing haemodynamic stability by administration of Calcium Gluconate (50 – 100 mg/kg) to correct hyperkalemia and hypocalcemia, Intravenous insulin (0.2 – 0.25 U/kg) to correct hyperkalemia, and sodium bicarbonate to correct metabolic acidosis. Fluid therapy is administered (Ringer's lactate – balanced polyionic solution).

Cystocentesis, either blind or ultrasound guided can be performed to relieve pressure in the bladder, to prevent the complication of urinary bladder rupture during catheterization.

Retrourohydropulsion can be done to push the calculi into the urinary bladder, thereby removing the calculus in the urethra and restoring normal urination.

The various catheters that can be used include Tom Cat Catheter, Minnesota olive tipped catheters, red rubber catheter, foley's catheter etc.

Voiding urohydropulsion can also be performed, wherein the animal is sedated and lifter to stand on two hindlimbs. The urinary bladder is compressed and the urine voided forcefully along with the calculus can be collected for further analysis.

Surgical procedures like urethrostomy and cystostomy can be performed should there be any necrosis of the tissues surrounding the obstruction site due to ischemia and trauma.

Further therapy can be provided by monitoring urine output and monitoring the electrolyte levels, blood pressure, Prazosin and phenoxybenzamine to relieve urethral spasms and relax urethra, ECG monitoring, administration of antibiotics (if there is any evidence of infection) and pain management (No use of NSAIDs due to recent renal insult)

Uroperitoneum

Uroperitoneum is defined as the damage to the integrity of the lower urinary tract (Bladder and urethra). There is leakage of urine into the abdomen. This can be due to trauma, neoplasia, prolonged obstruction and overzealous placement of urinary catheters. It should not be confused with uroperitoneum, which is the damage to the integrity of the upper urinary tract (Kidneys and ureters).

Creatinine, an osmotically active particle, present in the urine, leaks into the abdominal cavity, and causes water to be drawn into the abdominal cavity, thereby causing volume depletion and haemodynamic compromise.

The clinical signs associated with uroperitoneum include abdominal distension, abdominal pain, palpable fluid wave in the abdomen, palpable bladder (if urine is slowly oozing into the abdomen), and blood tests can reveal metabolic acidosis, azotemia and hyperkalemia.

In dogs, the abdominal fluid creatinine: serum creatinine ratio greater than 2:1 and the abdominal fluid potassium: serum potassium ratio greater than 1.4:1 is highly suggestive of uroperitoneum. In cats, the abdominal fluid creatinine: serum creatinine ratio greater than 2:1 and the abdominal fluid potassium: serum potassium ratio greater than 1.9:1 is highly suggestive of uroperitoneum.

The level of urea in the abdominal cavity is not of importance as it gets rapidly diffused into membranes for equilibrium.

Diagnostic imaging techniques include plain radiography (bladder wall not visualized, fluid shadow in the abdomen), contrast radiography (determine the site of obstruction), excretory urography (to check patency of the kidneys, ureters, bladder and urethra), retrograde urethrocystogram (to check the patency of the urinary bladder and urethra) and ultrasonography (to visualize the real time oozing of fluid) from the bladder into the abdomen.

Treatment is aimed at correction of acid base disturbances and electrolyte imbalances, fix a urinary catheter to keep the bladder decompressed, placement of a peritoneal dialysis catheter to remove the abdominal effusion fluid, and surgical repair for proper integrity of the urinary tract.

Acute Renal Failure

Acute renal failure, now known as acute kidney injury (AKI), is a sudden decrease in renal function due to toxicants in the blood, diseases causing renal insult, etc. Normally, 20 – 25% of the total blood flow reaches the kidneys. Out of the renal blood flow, more than 90% reaches the renal cortex and only less than 10% reaches the renal medulla, thus making the renal medulla more susceptible to ischemic injuries and the cortex more vulnerable to the toxins and their effects.

Concentration of toxins in the renal cortex, hypotension and hence ischemia, thereby compromising the oxygen and energy supply, are important factors in the pathophysiology of Acute Kidney Injury.

Acute Kidney Injury might also progress to chronic kidney disease in untreated cases.

AKI Grade	Blood Creatinine	Clinical Description
Grade I	<1.6 mg/dl (<140 µmol/l)	Nonazotemic AKI: a. Documented AKI: (historical, clinical, laboratory, or imaging evidence of AKI, clinical oliguria/anuria, volume responsiveness‡) and/or b. Progressive nonazotemic increase in blood creatinine: ≥ 0.3 mg/dl (≥ 26.4 µmol/l) within 48 h c. Measured oliguria (<1 ml/kg/h)# or anuria over 6 h
Grade II	1.7 – 2.5 mg/dl (141 – 220 µmol/l)	Mild AKI: a. Documented AKI and static or progressive azotemia b. Progressive azotemic: increase in blood creatinine; ≥ 0.3 mg/dl ≥ 26.4 µmol/l) within 48 h),or volume responsiveness‡ c. Measured oliguria (<1 ml/kg/h)# or anuria over 6 h
Grade III	2.6 – 5.0 mg/dl (221 – 439µmol/l)	
Grade IV	5.1 – 10.0 mg/dl (440 – 880 µmol/l)	Moderate to Severe AKI: a. Documented AKI and increasing severities of azotemia and functional renal failure
Grade V	>10.0 mg/dl (>880 µmol/l)	

(Courtesy: International Renal Interest Society, 2023)

The International Renal Interest Society has provided AKI Grading Criteria and divided the disease into five grades based on serum creatinine levels and urine production levels.

Acute Kidney Injury can also be classified based on the RIFLE system, where R – Risk, I – Injury, F – Failure, L – Loss and E – End stage.

RIFLE staging system

	Urine Output	Serum Creatinine
Risk	Decrease in GFR ≥25%; <0.5 mL/kg/h for ≥6 h	≥1.5-fold increase from baseline serum creatinine
Injury	Decrease in GFR ≥50%; <0.5 mL/kg/h for ≥12 h	≥2.0-fold increase from baseline serum creatinine
Failure	Decrease in GFR ≥75%; <0.3 mL/kg/h for ≥24 h or anuria ≥12 h	≥3.0-fold increase from baseline serum creatinine; or an absolute serum creatinine ≥354 μmol/L (4.0 mg/dL) with an acute increase ≥44 μmol/L (0.5 mg/dL)
Loss	Persistent acute renal failure: complete loss of kidney function for >4 wk	
End stage	Complete loss of kidney function for >3 mo	

Acute Kidney Injury can also be classified based on the AKI network staging system.

AKIN staging system

	Urine Output	Serum Creatinine
Stage 1	<0.5 mL/kg/h for ≥6 h	≥26.5 μmol/L (0.3 mg/dL) or ≥150%–200% increase from baseline serum creatinine
Stage 2	<0.5 mL/kg/h for ≥12 h	>200%–299% increase from baseline serum creatinine
Stage 3	<0.3 mL/kg/h for ≥24 h or anuria ≥12 h	≥300% increase from baseline serum creatinine or absolute serum creatinine ≥354 μmol/L (4.0 mg/dL) with an acute increase of ≥44 μmol/L (0.5 mg/dL)

Acute Kidney Injury an also be classified based on AKI network system.

VAKI staging system

Stage 0	Creatinine increase <150% from baseline
Stage 1	Creatinine increase of 150%–199% from baseline, or: Creatinine increase of 26.5 μmol/L (0.3 mg/dL) from baseline
Stage 2	Creatinine increase of 200%–299% from baseline
Stage 3	Creatinine increase of ≥300% from baseline or an absolute creatinine value >354 μmol/L (4.0 mg/dL)

The clinical signs are non specific, and include Lethargy, depression, anorexia, vomiting and diarrhoea. There will not be weight loss. Large painful kidneys can also be palpated on examination.

Blood Urea Nitrogen and Creatinine will be elevated due to azotemia. Azotemia can be prerenal (due to dehydration, hypotension, toxin etc.), renal azotemia (due to sepsis, microthrombi, etc.) and post renal azotemia (due to obstruction).

Stress leukogram (neutrophilia, lymphopenia, monocytosis and eosinopenia) can also be evident in Acute Kidney Injury.

The Biomarkers for Acute Kidney injury include Interleukin – 18, Neutrophil gelatinase – associated lipoprotein, Kidney injury molecule – 1 and Liver Fatty Acid binding protein.

On ultrasonographical examination, the Left Kidney: Aorta ratio is greater than 9.1

Treatment is aimed at expanding vascular volume and facilitating diuresis. Fluid therpay can be initiated at a rate of 5 – 6 ml/kg/hr. Sodium Chloride (0.45%) is more preferred because it provides more free water. After volume repletion and becoming normotensive, the urine output should range between 1 – 2 ml/kg/hr.

Antibiotics (Ampicillin – 22 mg/kg IV q8h and enrofloxacin – 5 mg/kg IV q24h) can be administered if there is evidence of infection.

To control gastric acid secretions and ulcers, Ranitidine @ 2 mg/kg IV q8h, Famotidine @ 0.5 mg/kg IV q12h, or Sucralfate @ 0.5 – 1 g PO q6 – 12h can be provided.

To control vomiting, Metaclopramide @ 1 mg/kg per 24h IV CRI, or Chlorpromazine @ 0.2 – 0.5 mg/kg Im can be given. Chlorpromazine causes hypotension, and so it should not be administered when the animal is hypotensive.

Diuresis is facilitated by providing Isotonic crystalloid @ 10 – 20 ml/kg for 15 – 20 minutes, Frusemide @ 2 – 6 mg/kg q6-8h, Mannitol @ 0.5 – 1 g/kg in 20 minutes, Dextrose @ 25 – 50 ml/kg 0f 10 – 25% slution for 1 – 2 hours.

Administration of Dopamine is said to increase renal blood flow, but its use is controversial, especially in cats. Dopamine causes hypotension, and so it should not be administered when the animal is hypotensive.

Feline Urethral Obstruction

Definition

- A "blocked cat" refers to a male cat with urethral obstruction, leading to inability to urinate, a life-threatening emergency.
- Most commonly caused by urethral plugs, uroliths, or urethral spasms.
- Often associated with Feline Lower Urinary Tract Disease (FLUTD).

Etiologies

- Urethral plugs: Combination of struvite crystals, mucus, and proteinaceous material.
- Urolithiasis: Most commonly struvite and calcium oxalate stones.
- Urethral spasms or strictures.
- Idiopathic cystitis (most common underlying cause).
- Neoplasia (rare).

- Infections (less common, but should be ruled out).

Predispositions
- Neutered male cats (narrow urethra).
- Obese cats.
- Cats aged 2–6 years.
- Indoor cats with low activity levels.
- Dry food diets (low moisture content).
- Cats with a history of FLUTD.

Contributing/Aggravating Factors
- Stress (changes in environment, household, other pets).
- Low water intake.
- Lack of environmental enrichment.
- Cold weather (reduced activity and hydration).
- Multicat households (competition for resources).

Pathogenesis
1. Initial insult: Inflammation of bladder/urethra or crystal formation.
2. Urethral obstruction: Mechanical (plug, stone) or functional (spasm).
3. Urinary retention: Increased intravesical pressure.
4. Back pressure on kidneys: Leads to post-renal azotemia.
5. Hyperkalemia & acidosis: Due to decreased renal excretion → causes bradycardia and arrhythmias.
6. Uremia and bladder rupture may follow if untreated.

Clinical Signs
- Stranguria (straining to urinate).
- Pollakiuria (frequent attempts).
- Hematuria.
- Painful abdomen.
- Licking of the genital area.
- Vomiting.
- Lethargy.
- Collapse (in severe cases).
- Bradycardia (due to hyperkalemia).

Diagnostic Parameters and Techniques
Physical examination:
- Firm, painful, distended bladder.
- Penile tip may be red/inflamed.

Bloodwork
- Elevated BUN, creatinine, and phosphorus.
- Hyperkalemia and metabolic acidosis.

Urinalysis
Crystals, RBCs, WBCs, protein.

Imaging
- **Abdominal radiographs**: Detect uroliths.
- **Ultrasound**: Check bladder wall, stones, effusion (if rupture suspected).

ECG
Look for peaked T waves, bradycardia, AV block from hyperkalemia.

Tom Cat Catheterization (Procedure Overview)
Stabilization
- IV fluids to correct dehydration and hyperkalemia.
- ECG monitoring.

Sedation/Anesthesia
Use opioids + dexmedetomidine or propofol if severe.

Preparation
Clip and aseptically prepare penis and prepuce.

Catheterization
1. Use Tomcat catheter (rigid) or polyurethane/slippery Sam (flexible).
2. Gently insert and flush with sterile saline to dislodge plug.

Once patency achieved
- Replace with **soft indwelling catheter** and secure to tail.
- Attach to **closed urinary collection system**.

Post-placement care
- Monitor urine output (1–2 ml/kg/hr).
- Continue IV fluids.
- Monitor for recurrence of obstruction.

Anti-Stress Medications
Pheromone therapy
Feliway® diffusers (synthetic facial pheromones).

Anxiolytics
- Fluoxetine (SSRI).
- Amitriptyline (TCA, also reduces bladder inflammation).
- Alprazolam (short-term use).
- Gabapentin (for stress and pain control).

Pain Management
- **Opioids**: Buprenorphine, Methadone.
- **NSAIDs**: Meloxicam (only after rehydration and stable renal function).
- **Gabapentin**: Neuropathic pain.
- **Local anesthesia**: Lidocaine gel for catheterization.

Diet Management
Prescription urinary diets
Hill's c/d Multicare, Royal Canin Urinary SO, Purina UR.

Goals
- Dissolve struvite (if applicable).
- Reduce crystal formation.
- Increase water intake.
- Encourage canned/wet food to improve hydration.

Environment Management
Stress reduction
Minimize changes in routine/environment.

Litter box management
1 box per cat + 1 extra, cleaned daily.

Water availability
Flowing water (fountains), multiple bowls.

Environmental enrichment
- Toys, perches, scratching posts.
- Play sessions to reduce stress.

Quiet resting areas
Avoid conflict with other cats.

Drug	Class/Use	Dose (Cat)	Route	Notes
Buprenorphine	Opioid analgesic	0.01–0.02 mg/kg	IV/IM/SQ/BUCCAL	Preferred for pain; minimal sedation
Methadone	Opioid analgesic	0.1–0.3 mg/kg	IV/IM	Useful in severe pain cases
Gabapentin	Neuropathic pain/anxiolytic	5–10 mg/kg BID (pain), 50–100 mg/cat 1–2 hrs pre-stress	PO	Can be used for both pain and stress
Meloxicam	NSAID	0.05 mg/kg SID (initial), then 0.1 mg/kg every 48 hrs	PO/SC	Only after hydration and normal renal parameters
Fluoxetine	SSRI (anxiolytic)	0.5–1 mg/kg SID	PO	Long-term use, onset in 2–4 weeks
Amitriptyline	TCA (anxiolytic, anti-inflammatory)	2.5–10 mg/cat SID	PO	Off-label; caution in cardiac patients
Alprazolam	Benzodiazepine anxiolytic	0.125–0.25 mg/cat BID	PO	Short-acting; for acute stress
Diazepam	Muscle relaxant	0.2–0.5 mg/kg	IV	Use with caution; avoid PO in cats (hepatic necrosis risk)
Prazosin	Alpha-1 antagonist (urethral relaxant)	0.25–1 mg/cat SID–BID	PO	Helps relieve urethral spasm post-deobstruction
Phenoxybenzamine	Alpha blocker	0.5–1 mg/kg SID	PO	Delayed onset; less commonly used now
Acepromazine	Tranquilizer	0.01–0.05 mg/kg	IM/SQ/PO	Can aid in urethral relaxation, stress relief
Dantrolene	Muscle relaxant	0.5–2 mg/kg SID–BID	PO	Skeletal muscle relaxant (rarely used)
Feliway® pheromone	Synthetic pheromone	As directed (diffuser/spray)	Environmental	Reduces environmental stress; adjunct therapy

Conflict of Interest
Not available

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