Managing the Difficult Urethral Obstruction

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Overview and pathophysiology

Feline urethral obstruction is one of the most common emergency presentations in the cat, accounting for approximately 9% of feline emergency admissions.¹ While there are many factors that may play into the development of lower urinary tract diseases in the cat, matrix-crystalline plugs and urolithiasis are the most common causes of obstruction.² Cats with urethral obstruction may have signs localized to the lower urinary tract including dysuria, stranguria, pollakiuria, hematuria, vocalizing, and pain, or they may show signs of systemic illness such as vomiting, lethargy, or collapse. Cats with obstructive urinary tract diseases may or may not have demonstrated preceding signs of lower urinary tract disease.

Following the development of urethral obstruction, clinical signs of uremia typically develop within 24 hours.³ Dehydration occurs due to decreased water intake and ongoing fluid losses secondary to vomiting. Acid-base (metabolic acidosis) and electrolyte disturbances (hyperkalemia and hyperphosphatemia) develop due to impaired excretion. Accumulation of metabolic wastes leads to post renal azotemia. Bladder capacity is reached, leading to rising intravesicular pressure and subsequently falling glomerular filtration rate (GFR). Prolonged obstruction may result in intrinsic renal failure. Damage to the urothelium and detrusor muscle may also develop during this time. If left untreated, death secondary to cardiopulmonary failure or hyperkalemia may occur within 3-6 days. Damage to bladder mucosa or urethra may shorten survival times.³

Diagnosis of urethral obstruction

Diagnosis of urethral obstruction is generally made on the basis of history and physical exam findings. Abdominal palpation typically reveals a turgid, painful bladder, though in rare cases, the bladder may be moderate in size if the cat is presented to the veterinarian shortly after clinical signs develop. Blood and/or crystalline debris may be visualized at the urethral orifice. The presence of bradycardia frequently indicates hyperkalemia, and severe systemic signs in conjunction with free abdominal fluid should prompt consideration of bladder leakage or rupture. In contrast, cats that present with stranguria but appear systemically healthy and have palpably small bladders typically have non-obstructive lower urinary tract disease.

At the time of presentation, a peripheral IV catheter is placed and blood is collected for complete blood count, serum biochemistry panel, and venous blood gas/electrolyte panel. The blood gas/electrolyte panel is particularly helpful as it provides rapid information on parameters such as potassium concentration (as well as acid-base status and renal values) that may affect initial interventions. Electrocardiography can also be helpful in the initial evaluation of the patient with urethral obstruction. Early ECG changes suggestive of hyperkalemia include bradycardia, dampened P-waves, tented T-waves, and prolongation of the P-R interval. As hyperkalemia worsens, loss of P-waves (atrial standstill) and widening of the QRS complex may develop. Electrocardiographic changes typically do not develop until potassium levels are greater than 7 mEq/L, but there is a great deal of individual variation in terms of patient response to hyperkalemia. Metabolic acidosis, hyponatremia, and hypocalcemia may contribute to the likelihood of hyperkalemic cardiotoxicity.

Once the animal has been medically stabilized and deobstructed, urine is submitted for urinalysis and culture. Because crystalline and cellular composition of the urine may change over time, evaluation of a fresh, undiluted sample is preferred. Diagnostic imaging should be performed to rule out cystic or urethral calculi. If a urolith or crystalline-matrix plug is retrieved at the time of deobstruction, composition should be determined as this may impact future therapies.

If free abdominal fluid is identified, fluid chemistry may be helpful in determining whether urinary tract rupture has occurred. An abdominal fluid:serum creatinine ratio of 2:1, or abdominal fluid:serum potassium ratio of 1.9:1 (cat) or 1.4:1 (dog) is predictive of uroperitoneum.⁴ Cytology of the fluid sample should also be performed to rule out urosepsis. Contrast cystourethrography is used to determine location and severity of the rupture.

Treatment of urethral obstruction

Fluid therapy

Initial management of urethral obstruction in the cat should focus on correction of hypovolemia, hyperkalemia, and other acid-base and electrolyte disturbances. In most cases, appropriate fluid therapy followed by restoration of urine flow will effectively correct these abnormalities. A peripheral IV catheter should be placed and fluid therapy instituted immediately using 0.9% sodium chloride or balanced electrolyte solution such as lactated Ringer's solution (LRS). A shock rate of fluids (66 ml/kg/hour in the cat) is calculated and then administered *to effect* in increments of approximately ¼ of the calculated dose, reassessing major body systems after each bolus. For example, the calculated shock rate in a 5 kg cat is approximately 330 ml, and should be administered in individual boluses of 50-100 ml every 10-15 minutes until cardiovascular status is restored. The goal of fluid therapy should be normalization of vital signs such as heart rate, level of consciousness, pulse quality, blood pressure, and capillary refill time. The specific type of intravenous

fluid selected is of lesser importance than the administration of appropriate volume. Although 0.9% sodium chloride has traditionally been selected due to its lack of potassium, studies in both experimental and clinical cases have shown that potassium containing solutions (LRS, Normosol-R) do not adversely affect the rate of resolution of hyperkalemia in cats with urethral obstruction when compared with 0.9% saline.^{5,6} Additionally, the buffered solutions are more efficient at restoring electrolyte and acid-base balance in severely affected animals.

Hyperkalemia

Relative or absolute bradycardia should be immediately investigated by monitoring electrocardiography and serum electrolyte concentrations. Severe electrocardiographic changes such as atrial standstill, widened QRS complexes, or sine wave formation provide strong indication for the administration of calcium gluconate. Calcium gluconate (10%) is given *slowly* at a dose of 0.5-1.5 ml/kg IV while carefully watching the patient's ECG for arrhythmias. Although calcium gluconate does not lower the serum potassium level, it has the immediate effect of buffering the myocardium from the toxic effects of hyperkalemia by restoring the normal difference between resting and threshold membrane potentials. Other intermediate to long-term interventions for hyperkalemia include the administration of regular insulin/dextrose and sodium bicarbonate, though these therapies are rarely warranted in animals with urethral obstruction as fluid therapy followed by timely restoration of urine flow are generally effective at reversing the hyperkalemia. However, if needed, 50% dextrose may be diluted 1:1 with saline and given at a dose of 1 gm/kg body weight to promote endogenous insulin release with subsequent potassium uptake by the cells through stimulation of sodium-potassium pumps. If regular insulin is used, it should be given at a rate of 1 unit insulin per 3 gm dextrose, though this is generally unnecessary and creates the need for careful blood glucose monitoring thereafter to avoid hypoglycemia. Sodium bicarbonate may also be given at a dose of 1 mEq/kg intravenously to facilitate intracellular potassium shifting in exchange for hydrogen ions.

Techniques for urethral deobstruction

During the initial exam, the urethra may be gently massaged, followed by careful palpation of the bladder to potentially dislodge superficial plugs. Extreme care should be taken to avoid accidental bladder rupture. While this technique is rarely effective, it is a simple extension of the initial physical exam and therefore may be worth trying in less severely affected cats prior to catheter deobstruction.

Although severely depressed patients may be deobstructed without the need for chemical restraint, sedation/analgesia is employed in the majority of "blocked" cats to improve patient comfort, facilitate deobstruction, and avoid urethral or bladder trauma secondary to patient struggling. Ketamine (100 mg/ml) may be combined with diazepam (5 mg/ml) in equal parts by volume and given at a dose of 1 ml/10 kg of the 50:50 mix. However, this combination should be avoided in cats with known or suspected hypertrophic cardiomyopathy, or when an undiagnosed murmur or gallop rhythm is present. In these cases, hydromorphone (0.05 mg/kg) in combination with diazepam (0.2 mg/kg) may provide a safer option.

Following sedation, the cat is positioned in dorsal recumbency with the legs pulled forward over the head. In this position, the prepuce may be retracted and the penis extruded by simply pushing the prepuce downward towards the anus. A further advantage to this technique is that it allows the urethra to be maximally straightened to facilitate deobstruction. The author's preferred technique for deobstruction uses an olive tip catheter (FUS needle 21 g x 1", Jorgensen Laboratories, Loveland, CO). This is a metal, bulb-tipped catheter that can be used to flush the urethra and either break down matrix-crystalline plugs or hydropulse them atraumatically into the bladder. Initially, the olive tip catheter is lubricated and inserted gently into the urethra to the site of the obstruction, approximately 1-2 cm. A 3 cc syringe is then used to lavage and break down the plug. Bits of the plug will often be seen emerging from the urethral orifice during the lavage. When the catheter is withdrawn, a strong stream of urine will frequently force the remainder of the plug from the urethra. Gentle bladder palpation may be used at this point to assist in the expulsion of the plug. To avoid urethral trauma, the catheter should not be forced past the obstruction. Instead, the lavage solution should be allowed to do the work. Additionally, acidic solutions should not be used for lavage as these have not been shown to be effective at plug dissolution and may further traumatize the urethral mucosa. If lavage alone is not successful at dislodging the urethral plug, the tip of the urethra can be pinched around the bulb tip of the catheter and hydropulsion used to push the plug back into the bladder.

Many clinicians use polypropylene "tomcat" catheters for the purposes of unblocking cats. These have the potential to cause additional trauma to the urethra when the rigid catheter is forced past the site of obstruction. If used, a number of steps may help to minimize iatrogenic urethral damage and maximize chances of success. (1) Completely straighten the urethra by pushing the prepuce dorsally towards to anus until the penis is parallel to the spine. (2) Use copious amounts of lubrication. (3) Hydropulse with sterile saline prior to advancing the catheter to assist in dislodging the plug. (4) Use a very light touch when advancing the catheter. Hold the catheter between index finger and thumb and twirl gently while advancing. Think about "picking a lock" when attempting to advance the catheter. Use finesse instead of force. (5) Once the catheter is well seated in the urethra, the penis may be allowed to retract into the prepuce. The prepuce may then be pulled caudally (toward tail tip) to further straighten the urethra while the catheter is advanced.

Some experienced clinicians advocate the use of cystocentesis prior to deobstruction to decompress the bladder and to potentially facilitate hydropulsion of urethral plugs. The author prefers to reserve this technique for use only as a last resort due to the number of

cats presenting to the emergency service with uroperitoneum and apical bladder tears following cystocentesis of overdistended bladders. However, it should be noted that our institution may see a biased population of more severely affected animals.

Cats that are critically ill, and those demonstrating large amounts of "sandy" crystalline debris in the urine, blood clots, uroliths, plugs hydropulsed into the bladder, bladder atony, or urethral narrowing are particularly at risk for reobstruction post-unblocking. For this reason, a soft, indwelling, 3.5-5 French red rubber catheter is placed following deobstruction to facilitate urine drainage overnight and to assist in quantitation of urine output. Indwelling catheters should be placed using liberal clipping and scrubbing of the perineum and aseptic technique to minimize risk of catheter-induced urinary tract infection. The tip of the catheter should sit just past the bladder neck to reduce risk of kinking or knotting. The catheter should then be connected to a sterile, closed collection system. To decrease the likelihood of premature catheter removal, careful attention should be given to suture placement. A piece of butterfly tape is placed around the catheter and appositional sutures are placed *at the margin* of the butterfly tape to prevent kinking of the catheter. The catheter body is then taped to the tail. An Elizabethan collar should be placed prior to anesthetic recovery.

Hospital management

Fluid therapy

Following initial stabilization and correction of hypovolemia, fluid rates should be adjusted to account for remaining fluid deficits, daily maintenance requirements, and ongoing losses. Deficits can be estimated as follows based upon clinical signs of dehydration: mild (5-6%), moderate (7-8%), and severe (8-10%). Multiplying the estimated percent dehydration by body weight gives the fluid deficit, which may then be replaced over the next 24 hours. For example, a 5 kg cat estimated to be 8% dehydrated would have an estimated deficit of 400 ml. To this value must be added maintenance needs (approximately 60 ml/kg/day) and ongoing losses. Ongoing losses following "unblocking" result from post-obstructive diuresis and can be estimated most easily by quantitating urine output. Normal urine output is approximately 1-2 ml/kg/hour (5-10 ml/hour in the average 5 kg cat). Urine output in excess of this amount typically results from post-obstructive diuresis. During the first 24 hours of therapy, a fluid rate should be selected that accounts for these ongoing losses. In other words, the intravenous fluids administered should slightly exceed measured urinary losses.

Urine output is quantified every four hours. Inadequate urine production (<1 ml/kg/hr) indicates inadequate fluid administration or urinary catheter occlusion with debris. After troubleshooting the catheter, a fluid bolus followed by an increase in fluid rate is indicated if urine output remains low.

Fluid therapy is typically tapered over the next 24-36 hours. Daily monitoring of electrolytes and renal values should be performed to ensure that azotemia resolves and electrolytes normalize. Potassium supplementation may be required during post-obstructive diuresis should hypokalemia develop.

Urinary catheter care

Indwelling urinary catheters and tubing should be cleaned externally once daily with a dilute chlorhexidine solution. Gloves should be worn and aseptic technique used when handling the catheters to avoid nosocomial infection. Bladder palpation should be performed every 4-6 hours to ensure that the bladder remains decompressed. When moving the patient, the urine collection system tubing should be clamped and the bag held below the level of the patient to prevent retrograde flow of urine into the bladder.

To minimize likelihood of catheter-induced urethral irritation or urinary tract infection, catheters should be removed as soon as possible. For most cats, the catheter is removed within 48 hours, but the presence of excessive crystalline debris or blood clots in the urine may necessitate longer indwelling catheter duration to avoid reobstruction. Use of antibiotics during hospitalization is not recommended as this is unlikely to prevent catheter-related infection, but may contribute to antibiotic resistance of organisms protected by the catheter biofilm. Culture should be performed prior to catheter removal, with antibiotic therapy initiated as indicated based upon results of culture and sensitivity.

Following catheter removal, patients should be monitored for an additional 12-24 hours to ensure that the urethra remains patent. Cats will typically urinate small volumes frequently following catheter removal due to irritation resulting from obstruction and catheterization. Although they may appear to strain in the litterbox, the bladder should remain small on palpation. A progressively distending bladder post-catheter removal typically indicates reobstruction (firm bladder, difficult to express) or bladder atony (large, flaccid, expressible). Cats with suspected urethrospasm post catheter removal may benefit from a smooth muscle relaxant following catheter removal (prazocin 0.5 mg/cat q24h).

Pain management

Urinary obstruction and initial management are frequently associated with significant discomfort. In our practice, buprenorphine (0.01 mg/kg IV q6h) is commonly used to provide analgesia for the first 24-48 hours.

Long term management

Strategies for long-term prevention of recurrence focus primarily on environmental modification and dietary changes. Occasionally, pharmacologic intervention may be warranted. An ample number of litterboxes should be provided, particularly in multi-cat households, and litterboxes should be cleaned regularly to encourage more frequent use. Canned or moistened food may decrease frequency of lower urinary tract episodes by promoting a more dilute urine and increasing frequency of urination. Fresh water should

be available at all times. In cases where obstruction was caused by struvite-matrix plugs, an acidifying diet may be of benefit. Antibiotics, anti-inflammatories, and antispasmotics have not been associated with reduction in frequency of episodes and their routine use is not recommended.

Perineal urethrostomy

Perineal urethrostomy may be considered in cases where frequency of urethral obstruction is unacceptable despite appropriate medical management or when irreversible changes in the urethra (stricture, scarring, urolithiasis) cause recurrent or persistent obstruction. Perineal urethrostomy has been associated with significant short and long term complications including recurrent urinary tract infection and stricture, and as such should not be considered a first line recommendation for cats with urethral obstruction.

References

Lee JA, Drobatz KJ. Characterization of the clinical characteristics, electrolytes, acid-base, and renal parameters in male cats with urethral obstruction. J Vet Emerg Crit Care 2003;13:227-233.

Osborne CA, Kruger JP, Lulich JP, et al. Feline matrix-crystalline urethral plugs: A unifying hypothesis of causes. J Small Anim Prac 1992;33:172-177.

Bartges JW, Finco DR, Polzin DJ, et al.. Pathophysiology of Urethral Obstruction. Vet Clin North Am Sm Anim Prac 1996;26:255-264.

Aumann M, Worth LT, Drobatz, KJ. Uroperitoneum in cats: 26 cases (1986-1995). J Am Anim Hosp Assoc 1998;34:315-324.

Cunha MG, Freitas CG, Carregaro AB, et al. Renal and cardiorespiratory effects of treatment with lactated Ringer's solution or physiologic saline (0.9% NaCl) solution in cats with experimentally induced urethral obstruction. Am J Vet Res 2010;71:840-846.

Drobatz KJ, Cole SG. The influence of crystalloid type on acid-base and electrolyte status of cats with urethral obstruction. J Vet Emerg Crit Care 2008;18:355-361.