FELINE URETHRAL OBSTRUCTION
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Pathogenesis of Obstruction
Urethral obstruction (UO) is a potentially life-threatening manifestation of feline lower urinary tract disease. Given their long and narrow urethra, male cats are much more likely to develop obstruction. The presence of a physical obstruction, such as a calculus or urethral plug, is often thought to be responsible for occluding the urethral lumen. However, functional obstruction secondary to urethral spasm and edema from underlying idiopathic cystitis (IC) may play an equally important role. The pathogenesis of IC is still unclear, but it appears to be a sterile inflammatory process. There is evidence that the disease may be related to a stress-induced imbalance between the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis. This imbalance is thought to result in impaired blood flow and release of inflammatory mediators which cause edema, smooth muscle spasm, and pain within the lower urinary tract. Pain, in turn, can contribute to the escalation of urethral smooth muscle dysfunction and urethral inflammation, thereby creating a vicious cycle. These conditions, either independently or in conjunction with a physical obstruction such as a plug or a stone, are ultimately what lead to urethral obstruction in cats.

Pathophysiology of Obstruction
Complete obstruction of the urethra leads to buildup of urine and pressure within the urethra and urinary bladder, which can lead to necrosis and mucosal injury. Pressure within the urinary bladder is then transmitted to the kidneys, with subsequent reduction of glomerular filtration. Within 24 to 48 hours of obstruction, the kidney’s excretory ability ceases, resulting in an accumulation of blood urea nitrogen, creatinine, phosphorus, potassium, and hydrogen ion in the blood, which contributes to the clinical signs associated with UO.

Uremia can cause depression, nausea, vomiting, and anorexia. The combination of decreased intake and ongoing GI losses can result in dehydration and potential for hypovolemia. Severe hyperkalemia is considered to be the most life-threatening aspect of UO because of its effects on the cardiovascular system. Elevations in serum potassium affect electrical conduction through the heart by diminishing the rate of depolarization, resulting in bradycardia. If the serum potassium level gets high enough, electrical activity in the heart can cease altogether, resulting in asystole. Severe metabolic acidosis can lead to denaturing of proteins, enzymatic dysfunction, and catecholamine hyposensitivity. The end result can be cardiovascular instability and potential for arrest.

History and Clinical Signs
The classic history associated with UO involves a male cat that has been vocalizing and straining unproductively in the litter box. However, these signs might be difficult to distinguish from a cat with IC. Owners might report no evidence of urination, but cystitis cats will often urinate very small amounts frequently, and sometimes outside the litter box, making it difficult to know for sure. In addition, multiscat households can present a challenge for owners because it is difficult to keep track of whether or not the cat has been urinating. One distinguishing feature of UO versus cystitis is that affected cats start to show signs of systemic illness as the obstruction progresses. This could include vomiting, lethargy, anorexia, and abdominal pain, which progress to changes in mentation and lateral recumbency. These signs are nonspecific if UO is not suspected, so obstruction should be considered as a differential for any sick male cat!

Clinical signs can vary considerably depending on the stage at which the patient is presented. Patients that present early may not have any striking physical exam findings aside from a firm, distended urinary bladder. In the “healthy” blocked cat, this is the most definitive way to help distinguish between obstruction and cystitis (as cystitis should have a small, barely palpable bladder). If the obstruction has been present for more than 24 hours, the patient may be showing signs of systemic illness. Systemic signs could include dehydration, bradycardia, and hypothermia. The presence of bradycardia in male cats should always raise concern for hyperkalemia, as the normal stress response to hospital presentation should result in tachycardia (though cats in septic or cardiogenic shock can also demonstrate bradycardia). In fact, the combination of bradycardia (HR < 140) and hypothermia (T < 96.6°F) has been found to be 98% predictive of serum potassium level greater than 8 meq/L in cats with urethral obstruction.

Initial Diagnostics and Stabilization: “Sick” Blocked Cat
Presentation of the “sick” blocked cat warrants immediate medical attention. An IV catheter should be placed and initial blood samples for PCV/TS and blood gas or chemistry panel obtained, if possible. Fluid therapy should be started immediately to support vascular volume and help dilute serum potassium concentration, even if bladder decompression cannot be performed immediately. There has been some debate as to which fluid type is ideal (0.9%
NaCl or a balanced electrolyte solution) based on potassium content and acid-base effects. However, it has been demonstrated that fluid choice does not impact outcome (survival, length of stay) or rate of reduction in serum potassium levels. The type of fluid probably doesn’t matter as long as an adequate volume is administered (Table 1). Care must also be taken with the amount given, as a recent study has shown that cats with UO are at risk for volume overload, especially if the patient has underlying heart disease.

An ECG should be placed, even if the patient is not demonstrating bradycardia, to determine any effects of hyperkalemia. Classic ECG changes associated with hyperkalemia include prolonged P-R interval, diminished to absent P waves, widened QRS, and tall, tented T waves. As hyperkalemia worsens, ECG changes can progress to atrial standstill, ventricular fibrillation, or asystole. While de-obstruction and IV fluids will ultimately be the primary means of eliminating potassium, this process takes time. If the patient has significant bradycardia (HR < 140), then immediate intervention to protect the heart (calcium gluconate) and promote intracellular shift of potassium (regular insulin, dextrose, and/or sodium bicarbonate) should be employed (Table 1). If insulin is administered, it is important that dextrose also be given to prevent the development of hypoglycemia.

Though controversial, cystocentesis can also be a part of initial stabilization by allowing immediate relief of pressure within the urinary tract and more rapid resumption of glomerular filtration. This could be especially important when busy emergency receiving does not initially afford time to pass a urinary catheter. In addition, only minimal sedation is usually needed to perform cystocentesis, and a “pure” urine sample can be obtained for urinalysis or urine culture. Finally, relieving the back-pressure against the obstruction may make for easier passage of a urinary catheter. The major concern raised against performing cystocentesis in UO cats is the potential for tearing or rupture of a distended and friable bladder. A recently completed study has demonstrated that development of clinically significant abdominal effusion, as evidenced by abdominal ultrasound, occurs very uncommonly after cystocentesis and that this procedure can be safely performed.

**Urethral Catheterization**

Passage of a urinary catheter to relieve a physical obstruction is generally considered to be essential in the management of UO. In order optimize the likelihood of successful catheterization and minimize damage to the urethra, heavy sedation/analgesia or anesthesia is recommended. A combination of injectable agents (ketamine [5–10 mg/kg] and diazepam/midazolam [0.25–0.5 mg/kg]; buprenorphine [0.01–0.02 mg/kg] and acepromazine [0.03–0.05 mg/kg], +/- propofol [1–4 mg/kg, to effect]) or general inhalant anesthesia may be used in the stable patient. A combination of buprenorphine or methadone (0.2–0.3 mg/kg) and diazepam/midazolam are often sufficient in the unstable patient, depending on the degree of metabolic derangement. Vocalizing or movement during catheterization attempts is likely to be associated with significant urethral spasm and an increased risk of urethral trauma. Under these circumstances, higher doses or additional medications should be given. The perineal region should clipped, prepped, and draped in order to minimize risk of contamination. A sterile, open-ended, semi-rigid catheter (polypropylene or polyurethylene) can be used initially to relieve the obstruction. Creating a mixture of saline and sterile lubricant (5:1) as the flush solution can serve to deposit lubricant along the entire length of the urethra and potentially decrease urethral damage. Another helpful technique is to pull the prepuce caudally once the catheter is seeded in the penile urethra. This will straighten out the urethra, thus making passage of the catheter easier and less traumatic. Once the initial catheter is in place, the urinary bladder can be emptied and flushed. Given that a polypropylene catheter is rigid and can cause significant urethral irritation, it should be withdrawn and replaced by a softer indwelling catheter, which is sutured in place. It has recently been demonstrated that use of a 3.5 Fr urinary catheter may be associated with less risk of immediate re-obstruction when compared to 5 Fr. Once the patient has been stabilized and the obstruction relieved, it is important to obtain abdominal radiographs (even a single lateral view encompassing the entire lower urinary tract) to assess catheter placement as well as the presence of calculi.

**Post-Obstructive Care**

Fluid therapy and monitoring of urine output are important aspects of post-obstructive care. Patients that have had prolonged obstruction are at risk for a post-obstructive diuresis, which has been demonstrated to occur in 46% of block cats. This diuresis is thought to occur through a number of mechanisms and can potentially result in massive urine production. It is therefore very important to keep up with urinary losses in these patients, as dehydration and hypovolemia could result. This can be achieved by matching urine production with fluid rate (UOP/hours collected) for the first 24 hours or so. Do not fear the high fluid rates that might be required! Once the azotemia has resolved, it is reasonable to gradually reduce the fluid rate to avoid driving further diuresis. Another potential concern is for inadequate urine production (< 1 ml/kg/hr) after obstruction is relieved. This could occur as a result of obstruction in
the collection system or dehydration. True oliguria could occur as a result of progression to acute renal failure, but this appears to be very uncommon in urethral obstruction.

Another important aspect of post-obstructive care is analgesia and sedation. Cystitis and obstruction, in addition to urethral catheterization, are painful and could be associated with risk of re-obstruction. Buprenorphine (0.01–0.02 mg/kg) generally provides sufficient pain control and has the benefit of oral administration. Acepromazine (0.05 mg/kg IV/IM or 0.5 mg/kg p.o.) can provide adequate sedation to decrease stress and agitation, so long as the patient is stable. In addition, the α antagonist effects of acepromazine might promote urethral relaxation and decrease risk of re-obstruction once the urinary catheter is removed. Prazosin (0.25–0.5 mg p.o. q12–24) could also be used for this purpose but does not provide the sedative effects of acepromazine.

Another frequent consideration in the post-obstructive period is whether the patient should be placed onto antibiotics, either to address existing urinary tract infection (UTI) or to prevent development of UTI from having a catheter in place. It has been well documented that antibiotics do not prevent development of catheter-associated UTI. In addition, two recent studies have prospectively looked at the incidence of UTI at presentation and secondary to catheterization. Both studies found that none were culture-positive at presentation. Through the course of catheterization, one study found that 33% (6/18) developed urinary tract infections, while the other found only 13% (4/31). Given the low incidence, performing a urine culture and sensitivity at the time of catheter removal is recommended to determine if a UTI has been introduced. As there is significant potential for contamination during catheter removal, the practice of submitting the catheter tip for culture should be avoided.

Electrolytes and renal values should be monitored every 12 to 24 hours and should rapidly correct to normal. Typically, if there is no significant reduction in renal values within 24 hours, complications may have occurred (renal failure, uroabdomen, etc.). Hypokalemia can develop (especially in patients with post-obstructive diuresis), and potassium should be supplemented accordingly. The urinary catheter should remain in place until the cat is clinically improved, blood work has normalized, post-obstructive diuresis has resolved, and urine is free of major debris, clots, or plugs in order to help minimize the risk of immediate re-obstruction. Once the catheter has been removed, the cat should be observed for 12 to 24 hours to ensure effective spontaneous urination prior to discharge.

**Alternative Management Protocols**

Unfortunately, the ability to provide the optimal treatment course outlined above may be limited by owner financial constraints. In addition, there is evidence that UO is as much a functional obstruction (urethral edema and spasm) as a physical one (plug or stone). When traditional management is not an option, a protocol of pharmacological manipulation (standardized doses of acepromazine and buprenorphine), low stress environment, bladder expression, decompressive cystocentesis, and subcutaneous fluid as needed for up to four days has been shown to result in spontaneous urination without the need for catheterization. This less invasive approach may prove to be significantly less expensive, depending on individual clinic fee schedules, and may be used as an alternative to euthanasia. However, given its nature, this protocol it is not recommended for cats in need of emergency stabilization or with significant azotemia. Furthermore, it should not be used as an alternative to traditional management, as no direct comparison between the two has been made.

In some cases, financial limitations might preclude the ability to provide any hospitalized care. Under those circumstances it may be necessary to offer euthanasia, especially for severely affected patients. For patients presenting in the earlier stages of obstruction, it may be possible to treat on an outpatient basis, though this should be reserved as a last resort. One option would be to provide sedation and analgesia (acepromazine and buprenorphine) and bladder decompression through either passage of a urinary catheter or cystocentesis. Catheterization offers the benefit of removing any physical obstruction within the urethra but could also result in damage or irritation to the urethra. Cystocentesis would likely be less expensive to perform and less injurious to the urethra, but might only provide temporary relief if a physical obstruction is present. In either approach, the patient would be discharged with recommendations as outlined below in the hopes that continued analgesia and sedation will allow for spontaneous urination to occur. Aside from anecdotal reports and clinical experience, there is no evidence to support the merits of either of these approaches, nor is there information regarding the likelihood of success or recurrence. The client would have to be well informed of the potential for treatment failure, and follow-up phone calls to determine response would be strongly recommended.
At-Home Care
Depending on the underlying cause, there is an approximately 25% to 40% incidence of recurrence with UO. Given this potential, at-home care may be extremely important to help decrease the likelihood of re-obstruction either immediately or in the future. Continued analgesia and sedation after discharge can be helpful, with administration of acepromazine and buprenorphine for 5 to 7 days. For patients demonstrating significant straining/urethral spasm after catheter removal, it may be of benefit to also administer prazosin. Antibiotics should only be dispensed based on results of urine culture taken at the time of catheter removal. Other recommendations which have been made to help decrease the risk of re-obstruction include increasing water intake by switching to wet food, flavoring the water, or using a running-water bowl. Given the potential role that stress may play in the pathogenesis of this disease, environmental enrichment may also help (visit www.indoorcat.org).

Table 1. Emergency Dosing for Severe Hyperkalemia (All medications given intravenously)

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose</th>
<th>Rate of Administration</th>
<th>When to Administer</th>
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</thead>
<tbody>
<tr>
<td>Isotonic crystalloid</td>
<td>10–15 mL/kg</td>
<td>15–20 minutes</td>
<td>Shock</td>
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<tr>
<td></td>
<td>5–10 mL/kg/hr</td>
<td>Constant rate infusion</td>
<td>Initial stages</td>
</tr>
<tr>
<td>Calcium gluconate</td>
<td>50–150 mg/kg</td>
<td>5 minutes</td>
<td>Bradycardia, Major ECG changes</td>
</tr>
<tr>
<td>Regular insulin</td>
<td>1 unit</td>
<td>IV push</td>
<td>Needed Ca gluconate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Potassium &gt; 8 mEq/L</td>
</tr>
<tr>
<td>Dextrose</td>
<td>0.5 g/kg</td>
<td>3–5 minutes</td>
<td>Needed Ca gluconate</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Potassium &gt; 8 mEq/L</td>
</tr>
<tr>
<td>Sodium bicarbonate</td>
<td>1 mEq/L</td>
<td>5 minutes</td>
<td>Potassium &gt; 10 mEq/L</td>
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</tbody>
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Selected References