ANIMAL MEDICAL CENTER of SEATTLE

Urethally Obstructed Felines: A Quick Splash Review

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Urethral obstruction (UO) in cats is one of the most common emergencies presenting to the veterinarian. It can be life-threatening due to the rapid development of electrolyte disturbances (i.e. hyperkalemia), post-renal azotemia as well as acid-base disturbances which can quickly lead to cardiovascular and neurologic dysfunction.

Cats presenting with a urethral obstruction can have a normal mentation, although depending on the severity of the biochemical alterations, poor perfusion and a comatose mentation can also present. Tachycardia, bradycardia or normocardia can be present regardless of the level of potassium.

The most important aspect of a presenting urethral obstructed cat is hemodynamic stabilization, primarily focusing on the "ABCs" (airway, breathing, circulation). Some cats may present with vomiting secondary to uremic nausea leading to ptyalism, which may cause an upper airway obstruction, therefore clearing of the airways (i.e. suctioning of the airway) takes precedent. Placement of an IV peripheral catheter and initiation of a balanced isotonic replacement fluid (i.e. Plasmalyte-A, LRS, Norm-R) to replace perfusion deficits, such as hypotension, may be indicated. Acidic crystalloid solutions such as 0.9% NaCl is not recommended due to potential exacerbation of metabolic acidosis – although when compared in clinical trials, there was no difference on resolution of electrolyte and acid-base disturbances with either solution. Low volume resuscitation is implemented in those felines with a heart murmur or those with a higher risk for fluid overload (i.e. 3-5 ml/kg of a balanced crystalloid solution). The author starts with 5-10 ml/kg of a balanced crystalloid solution with re-evaluation of the patient prior to considering subsequent doses of crystalloids due to the negative effects of overzealous crystalloid administration: pulmonary edema, cardiac contractility, gastrointestinal edema and coagulopathy. Correction of poor perfusion parameters may reflect as normalization of perfusion parameters (mucous membrane color, capillary refill time, heart rate, and pulse quality), determination of jugular venous distensibility, normal blood pressure, improved hyperlactemia, adequate urine output, and improved mentation. Obtaining blood for an emergency laboratory database (PCV/TP, Electrolytes, venous blood gas) will alert the clinician to any abnormalities that may need to be treated immediately.

After hemodynamic stabilization has occurred, correction of hyperkalemia (that can be exacerbated by metabolic acidosis) is important due to its negative cardiac effects.

An ECG should always be evaluated in a UO cat in order to diagnose any dysrhythmias associated with hyperkalemia. ECG changes associated with hyperkalemia include tall T-waves, prolonged P-R interval, bradycardia, sinoventricular rhythm and asystole. It is important to remember that there is no set potassium level that predicts the presence of a dysrhythmia (Lee and Drobatz JVECC 2006). If a dysrhythmia is not present with an associated hyperkalemia, IV fluid diuresis is likely sufficient to correct the hyperkalemia. The definitive treatment is to reestablish urine flow and glomerular filtration rate by placement of a urinary catheter to divert/remove the urine. IV fluids dilute the potassium and lower the serum concentration as well as increasing excretion through the kidneys by increasing GFR. Otherwise, treatment consists of 0.2 units/kg regular insulin IV, followed by 2 g dextrose/unit insulin administered. Insulin will activate the Na^+/K^+ ATPase pump to move potassium into the cell (temporarily reducing hyperkalemia). Dextrose will stimulate endogenous insulin production. Calcium gluconate (10%) given slow IV over 10-15 minutes will help to protect the myocardial cells and decrease the threshold potential. Sodium bicarbonate helps to reduce the pH in the extracellular space by helping to move intracellular hydrogen ions out while moving extracellular potassium ions in – this helps to reduce acidosis. Sodium bicarbonate is rarely used due to its iatrogenic complications, although may need to be considered in those patients with a pH <7.0). Ultimately, unblocking is pursued (which is beyond the scope of this text).

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Polypropylene catheters (i.e. tomcat catheter) can be used (with caution) in the initial relief of the obstruction, but should not be left indwelling due to it being more reactive/irritating than other catheter materials. Other material such as polytetrafluoroethylene and polyurethane catheters have the benefit of being firm at room temperature and softening at warm body temperatures – allowing for a single catheterization attempt and potentially less urethral trauma. Polyvinyl catheters (i.e. red rubber) are softer and may be less irritating as well. After the cat is unblocked, a closed collection system should be attached to the urinary catheter using a sterile empty IV fluid bag and fluid administration set. Urine output is measured every 2-4 hours once the cat is rehydrated and must be at least 1-2 ml/kg/hour, otherwise fluid intake should be adjusted to match urine output with close monitoring for signs associated with fluid overload. There is no standardized length of time in which a urinary catheter needs to be in place. Anecdotally, the author removes the urinary catheter based on resolution of hematuria or resolution/stabilization of azotemia (if initially present) or after 24 hours of urinary catheter placement. Studies have suggested that longer duration of urinary catheterization may be associated with a lower probability of short-term recurrent UO in male cats (Eisenberg, Waldrop et al JAVMA 2013). Some studies have also favored the use of a 3.5 fr catheter, as a 5 fr catheter was associated with a rate of recurrent UO that was >2.5 times as high as that associated with a 3.5 fr catheter – this was independent of the alpha-adrenergic receptor antagonist received (Hetrick and Davidown JAVMA 2013). Use of antimicrobials is not recommended, as the incidence of acquired urinary tract infection appears to be very low especially when aseptic technique is implemented. Further investigation is still indicated in regards to the use of urethral relaxants in the management of UO cats.

Common complications associated with post-urethral obstruction relief include: hyperkalemia, dehydration, azotemia, post-obstructive diuresis (POD), hypokalemia, urethral damage/tear, detrusor atony, urethral hyperactivity, reobstruction, hypocalcemia, anemia (Beer and Drobatz JVECC 2016), hypothermia and hypotension. POD occurs due to urea diuresis, salt diuresis and water diuresis – this leads to the inability of the kidneys to reabsorb sodium and water secondary to the obstruction as well as leading to loss of the medullary gradient (promoting large water losses from the kidneys). POD can last up to 24-48 hours. Close monitoring of the urine output and IV fluid intake will help to manage a POD. It is important to remember that not matching the urine output is more critical to a POD cat than concerns in regards to fluid overload as inadequate fluid replacement can lead to dehydration and worsening of the azotemia.

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