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This review accompanies the relevant episode of the Cutting Edge veterinary podcast. In each episode of this podcast, 3rd year students in the University of Calgary's veterinary medicine program fill you in on the most up-to-date literature and evidence-based practices on topics that matter to you, the practising veterinarian.

Exploring Controversies in the Management of Feline Urethral Obstructions.

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Feline urethral obstruction (UO) is a life-threatening component of feline lower urinary tract disease (FLUTD), accounting for 1.5 – 10% of cases presented to teaching hospitals and emergency veterinary practices.[1,2] Male cats are predominantly featured in case presentations. This is due to anatomical distinctions, as their urethras are considerably longer and narrower, making them more susceptible to obstruction.[3] UO is most commonly caused by feline interstitial cystitis, but may also occur less frequently due to urolithiasis, urethral plugs, neoplasia, or urinary tract infections.[4] Classic presentation includes young to middle aged male cats with a distended painful bladder, and a history of stranguria, dysuria, and anorexia. Systemic signs including tachypnea, bradycardia, vomiting, or signs of shock are also possible depending on severity.[3,5]

Management of feline UO should begin with a minimum emergency database (MEDB) that includes PCV, TS, lactate, glucose, and BUN. It is important to assess electrolytes, and renal parameters, particularly potassium. Hyperkalemia often leads to arrhythmias like bradycardia, which is defined as heart rate < 180 beats per minute in a clinical scenario.[6] These patients are in critical condition and should be monitored with an electrocardiogram to assess for arrhythmias. There is a considerable risk of cardiopulmonary arrest, therefore, treatment options will be more aggressive, focusing on correcting hyperkalemia and maintaining cardiac stability.

Treatment for feline UO generally includes stabilization through the administration of intravenous fluids and correction of any hyperkalemia that is present. Depending on the practitioner, this can be achieved through a combination of dextrose, insulin, and sodium bicarbonate.[6] In addition, calcium gluconate is often administered for cardioprotective effects. Once treatment for electrolyte derangements is initiated a urinary catheter will be passed to relieve the obstruction. It is recommended that patients are hospitalized and monitored for urine output for several days following initial stabilization.[7] Early intervention and treatment is necessary due to acute loss of renal function and metabolic derangements, which can rapidly

lead to cardiovascular instability.[2] Despite the severe complications associated with urethral obstruction, early treatment often leads to positive outcomes with 91.5 - 94.2% of cats surviving to discharge.[5,8] Long term prognosis is generally considered fair to poor, due to recurrent obstruction and many owners electing to euthanize. In a study by Gerber et al., 2008, it was observed that 36% of cats encountered recurrent urethral obstruction within a two-year span after the initial presentation.[8] In contrast, a study by Segev et al., 2011 reported a lower recurrence rate, with only 24% of cases experiencing recurrent urethral obstruction in a two-year period.[5]

Numerous controversial discussions exist surrounding the optimal approach for addressing UO as well as treatment options to prevent recurrent obstructions. Three of the major controversies include decompressive cystocentesis prior to unblocking procedures, cystocentesis without pursuing unblocking, and the use of prazosin to decrease the incidence of recurrent urinary obstructions. The aim of this literature review is to explore these three controversies and make evidence-based recommendations that can be utilized by the emergency clinician or general practitioner. We will evaluate these controversies while considering that the recommended approach is highly dependent on the clinical scenario and initial presentation of the patient as stable or unstable.

Decompressive Cystocentesis Prior to Catheterization

The use of decompressive cystocentesis prior to unblocking cats is a topic of great debate in the management of feline UO cases. Decompressive cystocentesis refers to performing cystocentesis prior to urinary catheterization, to decrease the size of the bladder and reduce intraluminal pressure. This technique may be advantageous when clinicians encounter challenges passing a urinary catheter to relieve an obstruction; it can provide immediate relief and buy more time for catheter placement.[9] There are several proposed advantages to decompressive cystocentesis prior to unblocking. This includes reducing patient discomfort, increasing perfusion to the bladder wall, improving glomerular filtration rate by decreasing renal backflow pressures, and improving ease of catheter placement by decreasing intraluminal pressure within the bladder.[10,11,12] Despite these advantages, there are risks associated with performing decompressive cystocentesis prior to unblocking cats. Potential complications include bladder wall damage or rupture, uroabdomen due to urine leakage, or abdominal hemorrhage due to inadvertent puncture of abdominal vasculature.[12,13] Clinicians have varying opinions regarding the use of decompressive cystocentesis due to the advantages and complications associated with this procedure. This section will focus on outlining the current literature regarding case management, patient outcomes, and complications associated with decompressive cystocentesis.

A randomized, blinded control study compared the clinical outcomes of cats presenting with UO and treated with or without decompressive cystocentesis prior to urinary catheter placement.[14] Researchers found no significant difference in the amount of time to place a urinary catheter or ease of placing the urinary catheter between groups.[14] Additionally, there was no significant difference in the amount of time the cats were hospitalized for, or the rate of

recurrent obstruction between these groups.[14] Although there is anecdotal evidence that decompressive cystocentesis may improve the ease of catheter placement, this study did not find evidence for or against its use. In summary, based on the current literature there does not appear to be any significant differences in clinical outcomes of cats treated with decompressive cystocentesis prior to urinary catheterization.

The relationship between decompressive cystocentesis and the development of abdominal effusion has also been evaluated in studies.[13,14] Specifically, this was investigated by performing an abdominal ultrasound before and after the decompressive cystocentesis procedure to quantify effusion. It appears that there is no significant difference in the development of abdominal effusion between cats that receive decompressive cystocentesis and those that do not.[13,14] However, studies show that abdominal effusion is commonly noted at presentation, prior to decompressive cystocentesis and urinary catheterization.[13,14] This may be due to high pressure within the bladder obstructing lymphatic drainage and leading to inflammation, or from urine directly seeping through the bladder wall.[15] Overall, decompressive cystocentesis may in theory result in small punctures in the bladder leading to urine leakage; however, it appears that the development of clinically relevant abdominal effusion is rare.[16]

Additional complications of decompressive cystocentesis include inadvertent puncture of abdominal aorta leading to hemorrhage. This complication is rare, but the risk of encountering this complication can vary based on the clinicians level of experience.[16] In one study, abdominal hemorrhage was found in one patient after receiving decompressive cystocentesis.[14] It is unknown if the hemorrhage was directly caused by the decompressive cystocentesis, however, there was no evidence of hemorrhage prior to the procedure.[14] Therefore, although decompressive cystocentesis rarely causes abdominal hemorrhage, there still remains a risk. This is worth considering and warrants a discussion with clients prior to considering decompressive cystocentesis as a component of a treatment plan.

In summary, there is no strong evidence for or against decompressive cystocentesis. The use of this procedure should fall to the clinician's discretion. Decompressive cystocentesis may be an appropriate option to consider if patients initially present as too unstable for urinary catheter placement, or if there are other reasons for a delay in catheter placement.[14,16] Additionally, if clinicians are unable to pass a urinary catheter it may be reasonable to perform decompressive cystocentesis in an attempt to ease catheterization.[14] It is important to communicate potential complications with clients prior to performing decompressive cystocentesis.

Decompressive Cystocentesis as Sole Management Strategy

The recommended treatment plan for cats presenting with urethral obstruction includes stabilization, IV fluid administration, urinary catheter placement to relieve the obstruction, and hospitalization.[15] This treatment plan does not come without considerable expenses. With the cost of treatment in mind, owners may elect humane euthanasia. Gerber et al., 2008 demonstrated that 23% of cats presenting with feline UO were humanely euthanized.[8] For this

reason, there is incentive to summarize the literature for alternatives when financial constraints are present and/or if the standard of care management cannot be performed Decompressive cystocentesis without placement of a urinary catheter is another topic of debate in the management of feline UO. This section will discuss the clinical presentation parameters that indicate decompressive cystocentesis without urinary catheter placement in feline patients with UO. Clinicians may use this literature review to guide their treatment plan for UO patients with varying clinical presentation.

As previously described, the proposed benefits of decompressive cystocentesis are pain alleviation, decreasing renal backflow pressure, and improvement of bladder wall perfusion and glomerular filtration rate. The largest complications associated with decompressive cystocentesis include bladder wall damage and rupture, uroabdomen from urine leakage and hemorrhage due to inadvertent puncture of abdominal vasculature.[9]

Previously, decompressive cystocentesis has been used as an approach to ease the placement of urinary catheters. As discussed above by Reineke et al (2021), decompressive cystocentesis provided no difference in the ease of urinary catheter placement or the time associated with placing the catheter.[14] Alternatively, Cooper et al (2010), proposed a protocol that includes decompressive cystocentesis without the placement of a urinary catheter at any stage of management.[15] This protocol includes pharmacological treatment, decompressive cystocentesis, subcutaneous fluids and ensuring the cat is hospitalized in a low-stress environment. In the Cooper et al (2010) clinical trial, 11/14 cats were treated successfully with only 2/11 cats reblocking three weeks post-treatment.[15] Despite bladder rupture being a large complication associated with decompressive cystocentesis, post-mortem evaluation in 3/4 of the cats who failed the protocol did not reveal evidence of ruptured bladder. Instead, the cats who failed treatment presented with significantly higher serum creatinine levels than the treatment-successful cats. This indicates that this protocol may benefit cases in which patients presenting with mild/moderate increased creatinine values as opposed to unstable patients. In addition, Cooper et al (2010), excluded patients with abnormal clinical findings including significant bradycardia (<120 beats per minute), severe hyperkalemia (>8.0 mmol/L), severe metabolic acidosis, unresponsive mentation or radiographic evidence of cystic or urinary calculi.[15] This further reflects that the protocol is not currently recommended for patients in critical condition, which must be considered by practitioners when they perform their triage and MEDB upon presentation. Veterinarians may attempt this treatment plan if their patient meets the clinical criteria described above and if their practice has a dark, quiet room for the patient. For instance, feline-only practices may consider this treatment as there are no dogs in the clinic to increase the stress of the patient.

The literature reflects that standard of care treatment, including urinary catheterization and hospitalization, should be offered as first line treatments to patients presenting with UO. If this is declined or the client has financial concerns, decompressive cystocentesis without urinary catheter placement may be attempted. Client communication is critical prior to initiation of treatment to discuss the prognosis associated with this procedure. Patients presenting with no abnormal clinical findings have a good prognosis whereas patients with

abnormal clinical findings, such as significant bradycardia, severe hyperkalemia, severe metabolic acidosis or unresponsive mentation, have a poor to guarded prognosis. The aim of offering decompressive cystocentesis to clients is to reduce euthanasia rates due to financial constraints; however, if standard of care treatment can be performed it should be considered as the treatment of choice.

Use of Prazosin to Prevent Recurrent Urethral Obstruction

The short-term use of prazosin following UO was popularized around 2006, largely replacing the use of phenoxybenzamine.[17] Phenoxybenzamine and prazosin are alpha 1 adrenergic antagonists, that have been used to reduce urethral spasms by acting on the smooth muscle of the proximal urethra.[18] Urethral spasms are proposed to be a potential risk factor for developing recurrent UO. Use of prazosin results in a lower incidence of reblocking in comparison to phenoxybenzamine.[17] However, based on recent literature the use of prazosin is highly controversial. A study by Conway et al., (2022), demonstrated that prazosin use may actually increase rates of recurrent obstruction within the first 14 days.[19] Additionally, a study by Hanson et al (2021), indicated that prazosin did not reduce the recurrence of UO, but instead increased rates of recurrent UO within their study groups.[20] In this study 16/65 cats developed recurrent UO, 11 of which had been given prazosin, and the remaining were part of the placebo group. Another study by Reinkeke et al., (2017), illustrated that prazosin had no significant effect in preventing recurring UO following resolution of the initial blockage.[21] For example, the prazosin group had nearly identical recurrence of UO as the placebo group. Therefore, there is not an overwhelming amount of research to support the claim that prazosin reduces rates of recurrent UO. Further prospective, randomized, and blinded clinical studies are needed to determine the effect of prazosin on the rates of recurrent UO.

Other Considerations

Aside from the three major controversies discussed in this review, there are other topics of debate among clinicians and within the literature. There are several other factors which may influence recurrent UO in patients. One feature to consider is the size of catheter used.[17,18] Evidence suggests the ideal catheter size which should be used in unblocking the male cat is a 3.5 french. A larger catheter, such as a 5 french, has been linked to higher likelihood of reblocking within 24 hours of removal of the catheter. The highest risk periods of a male cat obstructing following catheter removal is the first 24 hours and the first 30 days.[17] It is also recommended that indwelling catheters be left in place for longer than 24 hours.[22,23] A study by Tsuruata et al., (2022) demonstrated that the odds of reblocking is 6 times greater in cats which had indwelling catheters placed for less than 24 hours compared to cats which had indwelling catheters for over 24 hours.[22] Overall, it appears that short indwelling catheter times are associated with increased risk of recurrent UO in felines.[23]

Conclusion

This review has explored major management strategies of urethral obstructions in male cats. We have discussed decompressive cystocentesis prior to urinary catheterization, the use of decompressive cystocentesis and reducing stress with no urinary catheterization in more stable cases, and the use of prazosin to prevent recurrent urethral obstructions. All of which are topics that seem to be in contention among practitioners. Clinicians must use clinical judgment in every case presented to them to determine if one of these strategies may benefit their patient. The main driving factor in deciding which approach to use is the presenting condition of the patient.

Unstable patients may benefit from decompressive cystocentesis prior to catheterization to mitigate pain, increase glomerular filtration rate, and improve bladder wall perfusion. Additionally, this technique may be valuable if the catheterization is delayed. Decompressive cystocentesis prior to catheterization is less valuable in stable patients, due to the inadvertent risks associated with the procedure and less pressing timelines for unblocking patients. Unstable patients are poor candidates for conservative treatment approaches such as the use of decompressive cystocentesis without catheterization. In contrast, stable patients may have good prognosis with this treatment plan. There is insufficient evidence to support the use of prazosin to prevent recurrent obstruction. Overall, there are no definitive recommendations surrounding these controversies in the literature. Therefore, it is critical that clinicians use clinical reasoning and clearly communicate treatment plan options to clients when making decisions.

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