Outcome of male cats managed for urethral obstruction with decompressive cystocentesis and urinary catheterization: 47 cats (2009–2012)

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Abstract

Objective – To characterize the duration of urinary catheterization, length of hospitalization, complications and clinical outcome in cats with urethral obstruction managed with decompressive cystocentesis and subsequent urinary catheterization.

Design – Retrospective, observational, descriptive study.

Setting – University teaching hospital.

Animals – Forty-seven client-owned male cats diagnosed with urethral obstruction.

Measurements and Main Results – The medical records of 47 cats diagnosed with urethral obstruction were reviewed. Treatment of all cats included decompressive cystocentesis, placement of an indwelling urinary catheter and hospitalization for a minimum of 6 hours. Collected data included signalment, body weight, body condition score, owner-reported clinical signs, duration of clinical signs, vital signs, and venous blood gas or chemistry values. Mean duration of urinary catheterization was 27.9 hours, median length of hospitalization was 40 hours, and survival to discharge was 91%. Of 34 cats that had survey abdominal radiographs, 56% (19/34) had loss of peritoneal detail consistent with abdominal effusion. No cat was diagnosed with a ruptured bladder during hospitalization.

Conclusions – Decompressive cystocentesis, in cats with urethral obstruction, followed by placement of an indwelling urinary catheter, did not result in a diagnosis of bladder rupture in any cat. The source of and clinical significance of the reported abdominal effusion is not known. Survival to discharge, duration of catheterization, and length of hospitalization were similar to previously reported populations.

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Introduction

Feline urethral obstruction is a common urologic emergency.[1] Idiopathic causes, feline idiopathic cystitis, urethral plugs, and uroliths are the most commonly reported causes of obstruction.[2–4] Diagnosis is made via physical examination findings such as a palpably firm, unexpressable urinary bladder, and behavioral demonstrations consistent with an inability to urinate.[1,2]

Common clinical signs reported by owners include stranguria, dysuria, vocalization, lethargy, anorexia, and excessive grooming of the perineum.[1] Azotemia, ionized hypocalcemia, and hyperkalemia are biochemical abnormalities that have been documented in cats with urethral obstruction.[1,5]

Medical management of urethral obstruction consists of reversing life threatening electrolyte disturbances, maintaining adequate tissue perfusion, minimizing visceral pain, and alleviating the urethral obstruction.[1,6,7] To assist correction of these abnormalities, decompressive cystocentesis, prior to urethral catheterization, has been advocated by some clinicians.[7–9] Benefits described include immediate relief of patient discomfort due to bladder overdistension and reduction of deleterious renal back pressure. Additionally, lowering intraluminal bladder pressure facilitates retropulsion

of urethral plugs or urethroliths and improves the ease of catheterization.[8,9] Procedures facilitating urethral catheterization are of particular benefit given the risk of iatrogenic urethral trauma encountered during treatment of obstructed cats.[10–12] However, decompressive cystocentesis during initial management of feline urethral obstruction has been discouraged by some authors who cite a potential risk of bladder rupture and development of uroperitoneum.[13]

The purpose of this study was to evaluate outcomes in cats diagnosed with urethral obstruction that were managed with decompressive cystocentesis and subsequent placement of an indwelling urinary catheter. Our hypothesis was that decompressive cystocentesis, when followed by urinary catheterization, would not result in bladder rupture, increased duration of catheterization, increased length hospitalization, or increased mortality when compared to previously reported populations treated without decompressive cystocentesis.

Materials and Methods

Cats admitted to the Veterinary Medical Center (VMC), University of Minnesota, between January 2009 and August 2012 for urethral obstruction were identified via a search of the University of Minnesota's medical record database. Study inclusion was limited to male cats diagnosed with urethral obstruction based on clinical signs and physical examination findings, including a palpably firm, unexpressable bladder. Cats that underwent treatment for urethral obstruction in the Emergency Service (ES), with a combination of decompressive cystocentesis, subsequent placement of an indwelling urinary catheter, and hospitalization for a minimum of 6 hours were included. Cases were excluded from the study if the medical record was incomplete or medical therapy for urethral obstruction, including decompressive cystocentesis or attempts at or successful placement of a urinary catheter, had been performed in the 48 hours prior to presentation. Cats that had undergone any surgery involving the urinary tract in the previous 14 days were also excluded.

Data retrieved from the medical record included signalment, body weight, body condition score, owner-reported clinical signs, duration of clinical signs, temperature, pulse, and respiration. Additional data collected included venous blood gas[a] or whole blood chemistry[b] results obtained by the ES. Due to the upper limit for analyzer output, when BUN was reported as >50 mmol/L (>140 mg/dL), the value 50 mmol/L (140 mmol/L) was used and when potassium was reported as >9 mmol/L, the value 9 mmol/L was used in statistical analysis. Results of aerobic urine culture and urinary tract imaging (including radiographs, ultrasound, or contrast radiography) performed during hospitalization were also recorded. If medical imaging was performed, the presence of peritoneal effusion was recorded. If effusion was noted, records were evaluated for results supporting a diagnosis of uroperitoneum (including serum-to-abdominal fluid creatinine ratio, serum-to-abdominal potassium ratio, contrast imaging studies, or confirmation of bladder rupture at surgery or necropsy). An attempt was made to identify time of decompressive cystocentesis in relation to imaging time. Additionally, duration of catheterization, length of hospitalization, and survival to discharge was recorded. Nonsurvivors were categorized as died versus euthanized. Finally, if the cat represented to the VMC within 72 hours of initial discharge, diagnosis of confirmed or suspected uroperitoneum was recorded.

In our hospital, decompressive cystocentesis is performed using a 22-guage, 1.5 inch needle attached serially to IV extension tubing, three-way stopcock, and a syringe. The bladder is isolated and immobilized via gentle manual palpation; abdominal ultrasound can be used to confirm bladder location. The needle is inserted percutaneously at a 45 degree angle into the ventral and caudal aspect of the bladder and urine is aspirated. If the cat is fractious and painful, analgesia or sedation should be administered prior to cystocentesis. Ideally, the majority of bladder contents are removed during a single centesis.

Statistical Analysis

All statistical analyses were performed using a commercially available software program.[c] The Kolmogorov–Smirnov test was used to test the data for normal distribution. Parametric data are presented as mean (standard deviation); nonparametric data are presented as median (range). Nominal data are presented as proportion (95% confidence interval).

Results

Forty-seven of 84 patient records initially identified met the inclusion criteria. Of the 37 excluded, 5 cats had a decompressive cystocentesis and 7 underwent urinary catheterization or attempts at catheterization in the 48 hours prior to presentation. One had a perineal urethrostomy performed within the preceding 14 days. Five cats did not have an indwelling urinary catheter placed, 2 cats had no decompressive cystocentesis performed, and 16 cats were not hospitalized for a minimum of 6 hours. One cat presented twice for urethral obstruction during the study period and only one visit was included for evaluation.

Signalment

Of the 47 cats included, the majority were castrated males (45/47) and 2 were intact males. Breeds included domestic shorthair, medium, and longhair (41/47) cats. There were 6 purebred cats: Burmese, (1), Siamese (1), Rex (1), Abyssinian (1), Norwegian Forest Cat (1), and Siberian (1). The median age was 6 years (range 2–22 y), mean body weight 5.8 kg (SD ±1.4), and median body condition score 6/9 (range 3–9).

History and Physical Examination

The duration of owner-reported clinical signs prior to presentation was recorded for 46/47 (97.8%) cats. Duration of signs were less than 12 hours in 15/46 (32.6%), 12–24 hours in 20/46 (43%), and longer than 24 hours in 11/46 (24%) cats. Reported signs included stranguria (33/47, 70%, CI 57%–83%), vocalization (28/47, 60%, CI 46%–74%), vomiting (18/47, 38%, CI 24%–52%), anorexia (14/47, 30%, CI 17%–43%), and excessive grooming of the perineum (9/47, 19%, CI 8%–30%). Rectal temperature, pulse, and respiratory rate were obtained at presentation for all cats. The median rectal temperature was 38.5°C (101.3°F) (range 34.1–40.8°C [93.4–105.4°F]). Twenty-one percent (10/47) of cats were hypothermic (<37.8°C, 100°F), 60% (28/47) were normothermic [range ≥37.8–≤39.2°C (≥100–≤102.5°F)], and 19% were hyperthermic (>39.2°C, 102.5°F). The median heart rate was 200/min (range 120–260/min) and the median respiratory rate was 40/min (range 20–120/min).

Clinical Pathology

Clinicopathologic values are summarized in Table 1. Fifty-nine percent of cats (27/46) had a BUN value above the normal reference interval and 28% (13/46) had a BUN >50 mmol/L (140 mg/dL). Forty-one percent (13/32) of cats had a creatinine value above the normal reference interval and 33% (15/46) had a potassium value above the normal interval range, with 11% (5/46) having a potassium value >9 mmol/L. Twenty-seven percent (8/30) of cats had an ionized calcium level below the normal reference interval.

Urine obtained via cystocentesis prior to urethral catheterization was submitted for aerobic culture in 32 cats. A positive urine culture was obtained from a single cat and identified *Escherichia coli*. The remaining urine cultures were negative. Diagnostic abdominocentesis was not performed on any cat evaluated in this study.

Imaging

Survey abdominal radiographs, complete abdominal ultrasound, and double contrast cystourethrogram were obtained in 34, 3, and 3 cats, respectively. All imaging procedures were reviewed by a board certified radiologist and a formal radiology report was generated. Of the 35 cats evaluated by medical imaging, uroliths were identified in 2 cats (1 via radiographs and 1 via ultrasound). Focal loss of peritoneal detail consistent with effusion was noted on the abdominal radiographs of 19 (56%, CI 39%–72%) cats. Of these 19 cats, 2 underwent subsequent abdominal ultrasound and abdominal effusion was confirmed in both. There was no evidence of peritoneal effusion in the third cat that had an abdominal ultrasound; this cat did not have survey radiographs performed. There was no evidence of urinary tract leakage noted for 3 cats that underwent double contrast cystourethrography. One of these cats had effusion noted on prior radiographs and ultrasound. The other 2 cats had no evidence of effusion noted on radiographs performed prior to cystourethrography.

The timing of radiographs with respect to decompressive cystocentesis was evaluated. For 20 of the 34 cases, survey radiographs identified a distended urinary bladder, suggestive of imaging prior to cystocentesis. However, documentation that cystocentesis had not been performed prior to radiographs was verified from the medical record for only of 2 of these cats. Of the 20 cats with confirmed or highly suspected imaging performed prior to cystocentesis, 11 had effusion, including 1 of which was known to have not undergone prior cystocentesis. Of the remaining 14 cats with radiographic evidence of a small urinary bladder, prior decompressive cystocentesis was documented in 10. Six of these 10 had abdominal effusion. Of the final 4 cats with a small bladder, 2 had radiographic evidence of abdominal effusion.

Therapy and Hospitalization

Due to the retrospective nature of this study, therapy was not standardized. In general, treatment in the Emergency Service included placement of an IV catheter, administration of analgesia and/or sedation, and decompressive cystocentesis followed by placement of an indwelling urinary catheter attached to a closed collection system. Method of urethral catheterization and indwelling catheter type utilized was at the discretion of the attending clinician; urethral catheterization was performed after decompressive cystocentesis in all cases. Forty-six cats (98%) received analgesia or sedation, although time of administration in relation to decompressive cystocentesis could not be identified. A red rubber urinary catheter[d] was placed in 27/47

Table 1: Clinicopathologic values[a,b]

Variable	N	Median	Range	Mean ± SD	Reference interval
Venous pH	14	7.33	7.002–7.4		7.25–7.40
Venous pCO$_2$ (mm Hg)	14		28.7–46.8	33.9 ± 5.3	33–51
Venous base excess (mmol/L)	14	−8.5	−22 to−5		−5 to +5
Venous bicarbonate (mmol/L)	14		9.2–20.6	17.2 ± 3.3	13–25
BUN	46				
mmol/L		15.17	7.14–50		5.4–12.14
mg/dL		42.5	20–>140		15–34
Creatinine	32				
μmol/L		778	79.6–1538.2		88.4–194.5
mg/dL		8.8	0.9–17.4		1.9–2.2
Ionized calcium	30				
mmol/L		1.3	0.73–1.35		1.2–1.33
mg/dL		5.15	2.9–5.4		4.8–5.3
Sodium (mmol/L)	46	150	128–163		
Potassium (mmol/L)	46	3.95	3.1–>9		2.9–4.2
Chloride (mmol/L)	46	123	102–140		100–111
Blood glucose	46				
mmol/L			2.6–19.91	10 ± 3.3	3.41–7.37
mg/dL			47–362	181.7 ± 60.3	62–134

cats (57%) and a slippery sam urethral catheter[e] was placed in 12/47 cats (26%). The type of urinary catheter placed could not be identified in 8 cats (17%). Duration of catheterization could be identified in 39/47 (83%) of cats. Mean duration of catheterization was 27.9 hours (SD ±12.1 h), 28.6 hours (SD ±11.9 h) for survivors. Median hospitalization time was 40 hours (range 10–138 h) for all cats, 41 hours (range 16–138 h) for survivors, and 24 hours (range 10–96) for nonsurvivors.

Outcome

Forty-three cats (91%, CI 84%–99%) survived to discharge. The remaining 4 cats were euthanized during their hospital stay. Two of these cats were euthanized due to urethral obstruction following removal of their urinary catheter. The third cat was suspected of having pyelonephritis based on a positive urine culture and persistent azotemia. The final cat had persistent azotemia and hyperkalemia following the initial 12 hours of hospitalization and was suspect of developing oliguric renal failure secondary to urethral obstruction. A necropsy was performed on this cat and abdominal effusion was noted; however, the effusion was not further analyzed and was not definitively identified as urine. No source of leakage within the urinary tract was identified grossly.

A cystotomy for removal of cystic calculi was performed in 2 cats during their hospitalization following decompressive cystocentesis. No evidence of gross leakage from the bladder was noted at the time of surgery in either cat. Two cats underwent perineal urethrostomy during hospitalization, 1 following reobstruction subsequent to urinary catheter removal and the other to treat a urethral stricture identified via contrast cystourethrogram. Both recovered uneventfully.

Of the 43 cats that survived to discharge, 3 (7%, CI 0%–15%) cats represented to the VMC within 72 hours of initial discharge. One of these cats was hospitalized for urethral obstruction and discharged without complication. The remaining 2 cats were evaluated for stranguria without urethral obstruction and managed as outpatients through the ES. Of the 3 cats evaluated in the 72 hours following initial discharge, none were identified in the medical record as having or suspected to have uroperitoneum. None of the remaining 40 cats were known to have been diagnosed with uroperitoneum following discharge.

Discussion

This retrospective study evaluated 47 male cats diagnosed with urethral obstruction that underwent decompressive cystocentesis prior to placement of an indwelling urinary catheter as part of their therapy. None of the cats evaluated were diagnosed with a ruptured bladder during their hospitalization. Signalment, duration of clinical signs, clinical signs reported, initial vital signs, and degree of biochemical derangements were similar in this population when compared to previously described cats with urethral obstruction.[1] In this study, duration of catheterization and length of hospitalization, 27.9 hours and 40 hours, respectively, were similar to a study by Lee et al (catheterization 24 hours, hospitalization 43.2 hours) that retrospectively evaluated 223 cats with urethral obstruction managed in an urban, university setting. Survival to discharge in

the current study was 91%, consistent with previously reported survival rates of 93.6%,[1] 91%,[3] and 92%.[5] Euthanasia or deaths as a consequence of urethral obstruction following discharge were not evaluated.

Medical therapy for urethral obstruction can include treatment with isotonic crystalloids, administration of analgesia, management of life threatening electrolyte derangements, and relief of the obstruction.[1,6,7] In practice, time from initial presentation to establishment of urethral patency likely varies due to multiple factors including procurement of client consent, patient stability, caseload, clinician experience, and staffing. Ongoing obstruction contributes to patient discomfort, bladder injury, decreased glomerular filtration, and impaired tubular function.[2] Decompressive cystocentesis is a technique that provides relief of bladder, ureteral, and intrarenal pressures, temporarily ameliorating the effects of urethral obstruction.[8,9] Additionally, decompressive cystocentesis decreases patient discomfort, provides time for patient stabilization, and allows collection of unadulterated urine samples for analysis.[9]

Iatrogenic injury to the urethra and bladder has been reported as a result of urinary catheterization of obstructed male cats. A prior retrospective evaluation of 29 cats requiring management of urethral rupture found that urinary catheter placement during treatment of urethral obstruction was the cause of injury in 24 cats.[11] In another study describing 15 cats requiring perineal urethrostomy, all cats evaluated had urethral trauma attributed to prior catheterization.[12] Additionally, iatrogenic urethral trauma and bladder rupture sustained during catheterization are documented causes of uroperitoneum in cats.[10] By lowering intraluminal bladder and urethral pressure, decompressive cystocentesis may facilitate retropulsion of urethral plugs or uroliths, thereby potentially decreasing the risk of iatrogenic bladder or urethral trauma.[8]

Of the 47 cats evaluated in this study, none were diagnosed with a urethral tear or bladder rupture during their hospitalization following decompressive cystocentesis. Additionally, no evidence of bladder leakage was identified in the cats that underwent cystourethrogram or cystotomy. Peritoneal effusion was noted during necropsy performed on 1 cat; however, fluid analysis was not performed. No gross defect within the bladder wall was identified although this does not exclude urine leakage as the source of the effusion. None of the cats evaluated required repair of a ruptured bladder or urethral tear, although 2 cats did undergo perineal urethrostomy during hospitalization. One required surgery as a result of a urethral stricture and the other due to reobstruction.

In previously published case reports, septic uroperitoneum[14] and hemoperitoneum[15] have each been reported as adverse events following diagnostic cystocentesis in dogs. However, only one study has evaluated the use of decompressive cystocentesis in the management of feline urethral obstruction. A protocol employed by Cooper et al[16] utilized serial decompressive cystocentesis in conjunction with pharmacologic and environmental management in 15 cats. During hospitalization, 3 cats were diagnosed with uroperitoneum and hemoperitoneum was identified in 1 cat.[16] This is in contrast to the findings of the current study in which 47 cats were evaluated and none were diagnosed with adverse events related to decompressive cystocentesis.

The difference in outcome reported here, compared to the study by Cooper et al,[16] may be explained by the placement of an indwelling urinary catheter. Catheterization following decompressive cystocentesis keeps bladder volumes small and intraluminal hydrostatic pressures low, potentially reducing the risk of urine leakage from the centesis site. In the normal bladder, a fibrin clot forms and the detrusor muscle spasms during initial phases of incision healing, presumptively creating a seal and minimizing leakage.[17] It is likely that similar responses prevent leakage following centesis. However, following prolonged overdistension of the bladder, the mucosa and detrusor muscle may become damaged.[17] In the study protocol described by Cooper et al,[16] because no urinary catheter was placed and no definitive relief of the obstruction was performed, bladder volumes and pressures may have increased between episodes of decompression, possibly predisposing those cats to uroperitoneum. Additionally, decompressive cystocentesis was performed a mean of 3 times during hospitalization, increasing the risk of iatrogenic bladder and intra-abdominal trauma.

Loss of peritoneal detail consistent with some degree of abdominal effusion was noted on 56% of survey abdominal radiographs evaluated in this study. However, subsequent collection and analysis of the effusion was not performed in any cat, presumptively due to perceived clinical insignificance, and the type of fluid contributing to the loss of peritoneal detail on radiographs was not identified. Leakage of urine from the centesis site may have been developed, leading to the effusion observed. This could be explained if the cats were inadequately sedated and moved during the procedure or excessive digital pressure was applied to immobilize the bladder. Necrosis and other forms of structural damage to the bladder wall will also prevent the bladder's ability to contain urine.[9] Additionally, if substantial time had elapsed between decompression and urinary catheter placement, this could have resulted in increased intraluminal bladder pressures and urine leakage. However, even if a small amount of urine leaked at the time of decompression, when followed with indwelling urinary catheterization and standard care, it did not appear to

affect outcome when compared to previously described populations. Additionally, no cat represented to the Emergency Service and was subsequently diagnosed with uroperitoneum in the 72 hours following initial discharge. Alternatively, the effusion may not have not been urine. Altered lymphatic drainage in the presence of elevated hydrostatic pressures has been suggested as one cause of perirenal effusion noted in patients with ureteral obstruction[18] and a similar mechanism could contribute to effusion secondary to urethral obstruction. Although other types of effusion, including hemorrhagic and septic effusion, could be possible, this seems unlikely given that no cat required surgical intervention and hospitalization time and survival to discharge were similar to previously reported populations.

In an effort to better characterize the significance of the observed effusion, the timing of radiographs with respect to decompressive cystocentesis was evaluated. Although decompressive cystocentesis and urinary catheterization were both performed in the Emergency Service, due to variation in clinician preference for procuring radiographs at different time points in initial treatment and variable documentation of this timing in the medical record, timing of the radiographs could only be definitively identified in 12 of 34 cases. Fifty-five percent (11/20) of cats with a distended urinary bladder, consistent with images having been obtained prior to decompression, had radiographic evidence of effusion. Fifty-seven percent (8/14) of cats with a small urinary bladder, consistent with images having been obtained following decompression, had radiographic evidence of effusion. Although the number of cases is too small to evaluate statistical significance, approximately half of the cats in each group had effusion on radiographs, regardless of decompression prior to or following imaging. This is similar to the prior study involving decompressive cystocentesis where all cats had radiographs performed following their initial decompression and 53% (8/15) had evidence of caudal abdominal effusion.[16] The question arises as to whether abdominal effusion, as a result of increased bladder intraluminal hydrostatic pressure, is a normal sequela to urethral obstruction or if it is a consequence of urine leakage following decompressive cystocentesis. A prospective study evaluating the presence of abdominal effusion in cats with urethral obstruction, prior to any intervention, is needed.

Limitations to this study include the retrospective nature, small sample size, and lack of a control population. Because decompressive cystocentesis is routinely employed in our hospital, a control population could not be identified. A retrospective or prospective evaluation of cats managed with and without decompressive cystocentesis is needed. Additional limitations include the lack of diagnostic abdominocentesis and subsequent

imaging for cats that had abdominal effusion on initial radiographs. Placement of an indwelling urinary catheter and IV fluid therapy could have masked signs of uroperitoneum during hospitalization in some cats. Finally, there was not definitive follow up during the 72 hours following discharge for all cats. Because some may have been evaluated by another veterinarian following discharge from the hospital, episodes of bladder rupture requiring intervention following decompressive cystocentesis may have been missed.

In conclusion, decompressive cystocentesis, when performed in conjunction with placement of an indwelling urinary catheter, did not result in clinically significant uroperitoneum requiring surgical intervention, increased duration of catheterization, increased length of hospitalization, or increased mortality in this population when compared to previously published data.[1,3,5] Decompressive cystocentesis may reduce patient discomfort, aid in patient stabilization, limit deleterious effects of obstruction, and facilitate retropulsion of urethral plugs and uroliths. A randomized prospective study comparing cats with urethral obstruction managed with and without decompressive cystocentesis would need to be performed to further evaluate any specific effects from this procedure.

Acknowledgments

Footnotes

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