Session 1:

Feline Ureteral Obstructions: Between a rock and a hard place

Introduction

Ureterolithiasis has been on the rise in our feline patients. Ureteroliths are the most common cause of ureteral obstruction in the cat, although strictures, blood clots, and neoplasia have been reported as well.

Pathophysiology of Ureteral Obstruction

The vast majority (>95%) of nephroliths and ureteroliths in cats are composed of calcium oxalate, or occasionally calcium phosphate. How calcium oxalate stones form is an area of avid research. Potential contributions include areas in the interstitium of renal papillae that progressively mineralize or ductal plugs that form a nidus of mineralization.[1] Certain bacteria in the gut, such as oxalobacter, may decrease the risk of calcium oxalate stone formation. The role of acidifying diets, which decrease the risk of struvite stone formation in cats, is unclear in the pathogenesis of oxalate stones. Some ureteroliths are composed of dried solidified blood, and may be a manifestation of unrecognized renal hematuria. Because the feline ureter has an inner diameter of 0.4 mm, even small stones or accumulations of debris may cause obstruction. About 30% of cats have a ureter that runs dorsal to the vena cava (circumcaval ureter). The point at which the ureter crosses over the vena cava (proximal ureter) is an area prone to obstruction.

Complete obstruction of the ureter increases the pressure proximal to the obstruction. This pressure is transmitted back to the renal pelvis, distal and proximal tubules, up to the glomerulus. When the back pressure in Bowman's capsule exceeds the glomerular capillary pressure, glomerular filtration decreases, leading to inadequate GFR to maintain normal creatinine and BUN. After an initial increase in renal perfusion from vasodilation, within hours there is intrarenal vasoconstriction, which both further decreases GFR in the obstructed kidney and shunts blood to the presumptively functional contralateral kidney. If the obstruction is relieved quickly (i.e, within a week), the disruption may be completely reversible. If the obstruction persists for over 4 weeks, the resulting inflammation will have caused irreversible tubulointerstitial fibrosis and atrophy. Unilateral obstruction should not cause azotemia, unless the contralateral kidney has decreased function or is simultaneously obstructed.

Epidemiology and Clinical Presentation

Ureterolithiasis most commonly affects middle age cats with a median age of 7 years at diagnosis, whereas azotemia from chronic tubulointerstitial nephritis ("classic CKD") is most common in geriatric cats. Although calcium oxalate bladder stones are marginally more common in male cats and Persian, Himalayan, and Domestic Longhair breeds, there is no documented gender or breed predilection for ureteroliths.

With acute obstruction of one kidney with a dysfunctional contralateral kidney (e.g., from previously unrecognized obstruction leading to fibrosis), the presenting complaint may be clinical signs of acute uremia (lethargy, anorexia, vomiting, etc.). Abdominal pain, a common and excruciating sign in people, may or may not be recognized in cats. Stranguria may be present despite the upper urinary tract localization. Renal asymmetry (big kidney little kidney) is a common manifestation and may be

recognized during a crisis or during routine examination. Incidental discovery of ureterolithiasis during imaging for other reasons is also possible, although about 50% of cats with urolithiasis have CKD.

Diagnosis

Azotemia is not expected if the contralateral kidney is functional. Diagnosis generally involves abdominal imaging. Abdominal radiographs are valuable to determine the location and number of stones, and whether any are in the urinary bladder or kidneys. Abdominal ultrasound is also useful to assess degree of dilation of the renal pelvis, ureteral dilation, and degree of atrophy of the renal cortex and medulla secondary to hydronephrosis. Both radiographs and ultrasound will tell different parts of the story so it is best to do both if possible.

If hydronephrosis is present, percutaneous puncture of the renal pelvis under ultrasound guidance for injection of a radiocontrast agent (antegrade pyelography) may determine if obstruction is partial or complete. CT imaging may provide more detail than radiography/ultrasonography. Excretory urography (intravenous pyelogram) may be unhelpful, as renal perfusion of the obstructed kidney is limited. Similarly renal scintigraphy may not provide prognostic information about the reversibility of the damage after alleviating the obstruction.

Treatment

Treatment decisions for the cat with ureteral obstruction can be challenging. Medical therapy alone has a poor long-term survival rate (66%)[2] and while several surgical options exist, the choice can be highly dependent on the surgeon's skill and experience with each of them. Each has their own complications, follow-up requirements and costs associated with them.

Medical Therapy – While less successful as definitive treatment for ureterolithiasis, "medical" therapy is often the first line of treatment tried. This consists of IV fluid diuresis, diuretics such as mannitol, and monitoring of urine output. In addition, pain medication and alpha antagonists such as prazosin are also administered to induce ureteral relaxation. The theory is to increase urine flow and "push" the stones caudally into the bladder. Usually this treatment is tried for 24-48 hours before moving on to another treatment option. If there is no movement of the stones in that time, they are unlikely to respond with further treatment. This method generally is only successful in about 20% of patients, and more likely to work in animals with small stones that are in the distal 1/3 of the ureter.[2] Careful attention must be paid to prevent over hydration during this forced diuresis. Monitoring of urine output, weight, and respiratory rate are important indicators of over hydration. Repeated imaging of the renal pelvis and monitoring of its dilation is also essential to determine if medical therapy is effective. If no improvement in the dilation is noted (or worsening of the dilation is seen) after 24-36 hours, surgical intervention is almost always necessary. It is very important to warn owners of this likelihood at the start of treatment so that their expectations and willingness to pursue surgery can be discussed. In some practices, particularly those with experience surgical support, medical therapy is skipped all together with the exception of stabilizing the patient, and surgical intervention is the primary treatment.[3]

Surgical Intervention – Surgery of the ureter can be tricky. The feline ureter is normally only about 0.3 – 0.4 mm in width, although it is frequently dilated in patients with ureteral obstruction. Still, the dilation may only mean it is 2 or 3 mm in diameter. Ureterotomy involves incising longitudinally over the stone and removing it. Unfortunately the feline ureteral obstructions often involve multiple stones in a single ureter which complicates this surgery. This procedure requires microsurgical skill and should only be undertaken by a trained surgeon. Complications include urine leakage, stricture, and edema leading to

obstruction. One report quoted a peri-operative mortality rate of as high as 21%[4], however, the degree of renal compromise and the skill of the surgeon are of significant impact in the outcome.[5]

Nephrostomy tubes have been used in the past as an emergent procedure to assist in renal decompression and stabilization of the patient. Due to the increased use of stents and ureteral bypass techniques, and the high complication rate (particularly uroabdomen, 25%) associated with placement of percutaneous nephrostomy tubes, there has been a movement away from this practice.[2]

Ureteral Stenting – Antegrade placement of a double-pigtail ureteral stent has become more common in the last 10-12 years. The stent may be placed in one or both obstructed ureters, across the obstruction. The obstructing ureterolith may be left in place, or a ureterotomy performed over the stone and the stent placed to reduce pressure on the site and allow for good urine flow. The stent will not only allow for urine to flow through an obstructed ureter, but it also is thought to induce ureteral dilation. The stent generally remains in place, unless it migrates or peri-implant cystitis develops which may necessitate removal. In humans, the stents are rarely left in place longer than 8-10 weeks, and with further investigation into the duration of obstruction relief that continues after the stent is removed, it is possible the veterinary world may move in this direction as well, however, the current recommendation is to leave the stent in place unless there is a complication. In one study, short-term complications have been reported to be < 10% with stents specifically designed for cats, and long-term complications were experienced by 33% of cats, most of them minor and relating to bladder inflammation from the pigtail portion of the stent in the bladder lumen.[6] Other reasons to remove the stent or exchange it include mineral encrustation, infection, and stent migration. Perioperative mortality rate has been reported as ~8%.[5] Subcutaneous Ureteral Bypass (SUB) – A SUB provides a bypass route for the urine from the renal pelvis to the bladder, completely going around the obstructed ureter. A nephrostomy tube is placed in the dilated renal pelvis and a cystostomy tube is placed in the apex of the bladder. The two are connected in the subcutaneous space with a port that has a needle access membrane for urine sampling and contrast administration. These have previously been considered a “salvage procedure” when a stent was unable to be passed by an obstruction, however, its use is becoming more widespread as an alternative to the traditional stent. Placement of a SUB in cats with circumcaval ureters may be a better option and less likely to reobstruct that placement of a stent.[7]

Follow-up in patients with SUB placement is more involved than in those with stents. The SUB must be flushed via the subcutaneous port on a regular basis to maintain patency. Clients must be informed of this commitment, and be willing to continue post-operative care.

Nephrectomy – This is generally only performed if the affected kidney is so hydronephrotic that it contributes only minimally to GFR and is a likely nidus for infection. Due to the long term potential for development of CKD in these usually middle-aged cats, it is important to preserve as much functioning renal tissue as possible.

A recent, as yet unpublished, review of the use of stents and SUBs to treat benign ureteral obstruction in dogs and cats indicated that stents were superior in dogs and SUBs were superior in cats (A. Berent, personal communication, 6/2018).

Prognosis

Median survival time after placement of ureteral stents has been documented at > 500 days and in those cats dying from chronic kidney disease after stenting, it is > 1200 days.[6] Long term complications associated with ureteral stents have been reported to occur in ~ 30% of cases, and include urinary tract

infection/pyelonephritis, stent migration, obstruction of the stents with calculi, and ureteritis secondary to the stent.[6] Removal or exchange of the stent in each of these, along with associated treatment (i.e. antibiotics in the case of the UTI) generally solves the problem. Importantly, many of the cats who have their stents removed because of one of these complications do not necessarily re-obstruct in that ureter. If this does occur, placement of a SUB is often the next step to bypass the ureter altogether.

In a study investigating predictors of long-term survival in cats with ureteral obstructions requiring either or stent or a SUB, only the BUN at presentation appeared to predict outcome.[8] In addition, those patients with a lower IRIS stage at discharge tended to continue with a lower stage of CKD long term.

Not surprisingly, the prognosis for each cat is highly dependent on the degree of residual damage to the affected kidney, as well as whether the contralateral kidney has been previously injured. Many of these cats develop chronic kidney disease and while they have improvement in azotemia over the short-term post treatment, they may never return to a normal creatinine and BUN. Close monitoring of these cats for additional obstruction, since they will continue to be 'stone formers' as well as for progression of renal disease is important lifelong. The impact of placing these cats on a diet formulated for prevention of calcium oxalate stones, and other such treatments, has not been studied, however, it would not be imprudent to consider it, just as though the cat were forming calcium oxalate stones in the bladder.

References

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