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FELINE URETERAL OBSTRUCTION—DIAGNOSIS AND MANAGEMENT

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Ureteral obstruction from calcium oxalate ureteroliths or non mineralized debris has become one of the most significant causes of renal disease and acute uremia in cats over the past 15-20 years. Although greater awareness and imaging techniques have contributed to its increased identification in recent years, there is little doubt that this represents a relatively (since 1990’s) new clinical entity in cats that still is not recognized in all parts of the world. The clinical presentation depends on the completeness and duration of the ureteral obstruction and the number of kidneys affected.

Acute Unilateral Ureteral Obstruction: Generally, unilateral ureteral obstruction is clinically silent, potentially transient, and not associated with azotemia. Evidence of unilateral obstruction may be detected in cats presented for signs of acute abdominal pain, erratic or elusive behavior, hematuria, or on routine abdominal imaging.

Acute Bilateral Ureteral Obstruction: This presentation is uncommon but associated with severe acute signs including bilaterally enlarged and painful kidneys, severe progressive azotemia, and oliguria or anuria. Prognosis is good if the obstructions are alleviated because renal mass is intact, but reoccurrence may be common.

“Big Kidney-Little Kidney” Syndrome: This common presentation is seen in cats with a past unilateral ureteral obstruction that has caused the kidney to progress to a fibrotic end-stage. Subsequently, the contralateral, hypertrophied kidney becomes acutely obstructed by a ureterolith causing further enlargement and characteristic renal asymmetry, abdominal pain, progressive severe azotemia, and variable urine production. Prognosis is good if the obstruction is alleviated because adequate renal mass persists, but reoccurrence in the big kidney is common.
Bilateral Chronic Kidney Disease with Concomitant Ureteral Obstruction: This presentation is common and carries the most guarded prognosis because even with resolution of the ureteral obstruction, global renal function is severely compromised, and there may be little capacity for renal repair. The underlying chronic kidney disease may be secondary to fibrotic changes from previous ureteral obstruction or other intrinsic kidney diseases which may predispose urolith formation. The sudden obstruction (unilateral or bilateral) causes decompensation of the tenuous residual renal function and promotes the onset of overt uremia.

The usual clinical presentation of ureteral obstruction is for signs of severe acute uremia which includes anorexia, depression, intermittent vomiting, uremic odor, lethargy, weight loss, and oligoanuria which can be differentiated easily from other causes of acute uremia. The syndrome may occur in cats of all ages, but appears more prevalent in cats older than 7 years of age. Affected cats may have a previous history of calcium oxalate urolithiasis or an entirely unremarkable history. The uremia is readily confirmed by serum chemistries documenting variable to profound azotemia, metabolic acidosis, hyperphosphatemia, and variable hyperkalemia. Cats may present with dehydration or life-threatening overhydration depending on management prior to presentation. On abdominal palpation, the kidneys are asymmetrically or symmetrically enlarged or small according to the presentations indicated above. They are characteristically resilient but firm, smooth or irregular, and painful on palpation. The bladder may or may not contain urine. Interestingly, many cats with ureteral obstruction and severe uremia often display a clinical demeanor that is considerably better than cats with similar azotemia from other etiologies. Many cats are aggressive and discontented due to pain associated with the condition.

The diagnosis of ureteral obstruction is made on the basis of physical findings and clinical suspicion and is confirmed by abdominal imaging. Survey radiography and ultrasonography are highly complementary imaging modalities and both are indicated in cat with acute uremia. Ultrasonography provides greater delineation of renal geometry, intrarenal architecture, parenchymal consistency, and outflow integrity than survey radiography, but its sensitivity is only 70% to 80% for the diagnosis of ureteral obstruction. Hydronephrosis and dilation of the proximal ureter are the principal ultrasonographic features of ureteral obstruction. However, clinical experience has demonstrated that these findings may not be demonstrated for up to 4-7 days following complete obstruction of the distal ureter (personal observations).

The increased availability and utility of ultrasonography have curtailed the routine use of survey radiography to image the urinary tract, and this trend has likely delayed the broader recognition of this condition over the past 5 to 10 years. Survey radiographs may predict ureteral obstruction in cats if careful scrutiny is directed to barely perceptible radiodensities in the retroperitoneal space, expansion of the retroperitoneal space, and the presence of marked renal asymmetry. In 20-30% of cats with ureteral obstruction, no discrete calcific material is identified with either ultrasound or survey radiographs. For these circumstances, and for cases with multiple sites of obstruction or stenosis, antegrade pyelography or computed tomography can be used to define the indication and location for surgical intervention. Antegrade pyelography incorporates ultrasound-guided pyelocentesis and antegrade injection of positive radiographic contrast media into the renal pelvis and ureter to delineate their size and the patency of the ureter. Antegrade pyelography can be performed in specialty practice and reliably confirms the presence, degree, and proximal location of ureteral obstruction in cats. Computed tomography with contrast is a less invasive than antegrade pyelography and has become the preferred imaging modality at specialty hospitals to confirm mineralized and nonmineralized uroliths and the differential patency.
of the ureters. This is especially useful if the cat has bilateral ureteroliths, but these facilities are not as readily available. At times, unilateral ureteral obstruction can be identified as an incidental finding when cats are imaged for unrelated or vague complaints of illness or hematuria.

The short-term management of acute ureteral obstruction is directed primarily at stabilizing the clinical consequences of the oligoanuria and uremia and secondarily to alleviate the obstruction and re-establish renal function. In 25% to 30% of cases, the obstruction (particularly if distal in location) can be induced to pass spontaneously if the cat’s uremia is sufficiently stable to delay surgical intervention for 2 to 4 days. In situations where immediate surgical intervention is contraindicated (severe hyperkalemia, fluid overload) or unavailable, renal replacement therapy with hemodialysis should be instituted to alleviate the uremia while attempting to get the obstruction to pass. Intermittent boluses of mannitol at 0.5 to 1.0 gm/kg daily (if anuric) or B.I.D. (if oliguric) for 1 to 3 days has been the most effective therapy to facilitate passage of the obstruction (personal observation). Ureteral relaxant agents like prazosin (drug of choice) or amilriptyline can be combined with mannitol, but none of these therapies has undergone careful prospective evaluation, and their use remains anecdotal.

Most cats require surgical intervention or minimally invasive interventions to alleviate the obstruction. Until recently, surgical relief of the obstruction by ureterotomy or ureteral transection and neoureterocystostomy were the only available options to correct the obstruction. Ureteral surgery, however, is associated with very high perioperative mortality and complication rates and should be performed by surgeons with extensive experience with ureteral and microsurgical techniques.

Within the past 4-5 years, the application of ureteral stenting by surgical or minimally invasive techniques has revolutionized the immediate and long-term management of ureteral obstruction in cats. Ureteral stents, which pass from the renal pelvis to the urinary bladder, have dramatically improved the ability to achieve short-term patency of the ureter even if obstructing urolith cannot be removed. Ureteral stenting eliminates the immediate post operative functional obstruction seen commonly for 3 to 5 days as a result of ureteral swelling and inflammation associated with surgical intervention. The stent also facilitates surgical closure of ureterotomies and potential for post operative leakage and ureteral stenosis. Ureteral stenting can be performed safely even in severely uremic cats in which life-threatening complications of hyperkalemia and overhydration have not developed or have been corrected. Early surgical intervention and stenting improves the outcome from an acute obstruction and may preclude expensive intervention with dialysis.

Ureteral stenting has become the mainstay for the management of recurrent ureteral obstruction in cats. Thirty to forty percent of cats recovering from an acute ureteral obstruction suffer recurrent obstructions in the same or contralateral ureter with 6 months. Although, some of these obstructions will pass spontaneously, a permanent ureteral stent will eliminate the requirement for multiple surgeries and lessen progressive renal damage attending recurrent obstructive events. We have now maintained “permanent” ureteral stents in cats with recurrent obstruction for up to 2 years without additional obstructive events and stabilized renal function.

After ureteral patency has been established by spontaneous passage of the obstructing urolith, surgical resolution, or ureteral stenting, therapeutic attention must be directed to prevention of future occurrences. Two
Dietary approaches have been proposed to reduce calcium oxalate recurrence with both approaches reliant on reduction of the relative supersaturation potential for calcium oxalate. One approach focuses on the controlled reduction of dietary substrates for calcium oxalate nucleation and urinary pH control to lower the relative supersaturation potential. The alternative approach purports to control the potential for spontaneous homogeneous crystallization of calcium oxalate through contrived urinary dilution with the addition of increased dietary salt and controlled urinary pH. The efficacy of both theoretically conceived approaches await controlled therapeutic trial in predisposed or affected cats, and neither approach can be endorsed explicitly or preferentially at this time.

Controversy has evolved over the potential risks of dietary salt to increased water consumption, urine formation, and urinary dilution. A similar concern can be expressed over wide-spread endorsement for the use of subcutaneous fluid therapy in cats with chronic kidney disease secondary to ureteral obstruction. At the foundation of the concern is the potential for salt loading to expand body fluid volume and promote systemic hypertension. We investigated this potential and when cats ate a 1.3% sodium diet (the level suggested for calcium oxalate urolith prevention), extracellular fluid volume increased significantly consistent with ECF volume expansion in response to dietary sodium. Further, feeding the high sodium diet produced a highly statistically significant increase in systolic, diastolic, and mean blood pressure compared to feeding a low sodium diet. The clinical significance of these short-term and modest changes in blood pressure remains unknown for cats with IRIS Stage I or II (or greater) chronic kidney disease but should promote caution for the use of dietary or parenteral salt.

An alternative approach to reduce the homogeneous crystallization potential for calcium oxalate urolith formation is to establish urinary dilution with dietary water. Most cats can be trained to consume (essentially drink) a diet appropriate for their IRIS CKD Stage that is diluted with sufficient water to produce a watery consistency. Cats at very high risk for recurrent ureteral obstruction that will not eat a watery diet should be fed a liquefied diet through an esophageal feeding tube. We have extensive years of experience with this feeding option and find it highly successful, completely acceptable to the cat and client, and effective at maintaining appropriate nutrition, appropriate hydration, and urinary dilution.

Ureteral obstruction is an increasingly recognized feline disease with profound immediate and long-term consequences which should be actively evaluated in uremic cats worldwide. It requires accurate and immediate recognition and a coordinated medical, dialytic, surgical, and dietary approach to save cats that are most severely affected.