Canine Calcium Oxalate Urolithiasis

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INTRODUCTION

Calcium oxalate urolithiasis is a fairly common problem in both the canine and feline patient, and over the past 30 years, the incidence of both canine and feline calcium oxalate uroliths has increased to be about as common as struvite uroliths.[2,3,10,14,15] Hypercalcemia increases the risk of calcium oxalate stones, but it is not a common presentation for canine or feline patients.[2,3,9,10,13,15] More commonly, the underlying cause of calcium oxalate urolith formation cannot be determined.[4,9,10,15] Surgical intervention is necessary if the patient presents with urinary obstruction, but non-surgical means of intervention can be attempted if the stones are small enough to pass through the urethra.[2,4,7,8,9,12,13,16,21] Since calcium oxalate uroliths cannot be medically dissolved, dietary management and serial monitoring is necessary to prevent recurrence of calcium oxalate stones.[2,3,8,9,10,13,15,20]

HISTORY AND PRESENTATION

Typical presentation of calcium oxalate uroliths are toy and small breed dogs that are on average between 5 and 11 years of age.[8,9,10,13] Calcium oxalate stones are more common in neutered male dogs, and there is a breed predisposition in Bichon Frise, Lhasa Apsos, miniature Schnauzers, Pomeranians, Shi Tzus, and Yorkshire terriers.[1,3,8,9,10,13] Typical clinical presentation includes urethral obstruction signs such as urinary incontinence, stranguria, pollakiuria, hematuria, dysuria, or more general signs such as persistent urinary tract infections, painful abdomen, depression, lethargy, vomiting, and anorexia.[2,4,7,12] If the stones are small enough, the patient may present with signs of urinary blockage, which is a medical and possible surgical emergency.[2,4,7.12,19] Urinalysis can show hematuria, crystaluria, with possible pyuria; commonly there will also be acidic urine because calcium oxalate stones grow best in acidic

urine, and there may also have hypercalciuria, hyperpoxaluria, and highly concentrated urine.[3,5,8,15]

PATHOPHYSIOLOGY

Calcium oxalate stones initially form due to urine supersaturation with calcium and oxalate in acidic and highly concentrated urine.[2,3,9,13,15,17,20] Hypercalcemia increases the risk of calcium oxalate uroliths, and hypercalcemia can be due to excessive GI absorption of calcium, impaired renal reabsorption of calcium, or excessive skeletal mobilization of calcium.[2,3,9,10,13,15] However, hypercalcemia is a rare finding in dogs rather than cats with calcium oxalate urolithiasis (4% vs 35%), and if dogs have hypercalcemia and calcium oxalate stones it is usually associated with primary hyperparathyroidism.[2,3,10] If stones form around a suture nidus, they are most commonly calcium oxalate stones, therefore it is important to use suture patterns that do not penetrate the bladder lumen when closing a cystotomy.[2,8,12,15,16,20] Calcium oxalate monohydrate stones are more common than calcium oxalate dihydrate stones.[8]

Metabolic acidosis can cause hypercalciuira through bone turnover which increases the serum ionized calcium which increases the amount of calcium excreted in the urine and decreases the renal tubular reabsorption of calcium.[2,3,9,14,15] Although it is intuitive to think that decreased dietary calcium would decrease the amount of calcium excreted in the urine, the opposite is actually true – this is due to the relationship between GI absorption of calcium and oxalate acid.[2,3,10] Dietary forms of these substances form non-absorbable calcium oxalate in intestinal lumen, so if calcium is reduced without a complimentary reduction of oxalic acid, there is increased oxalic acid absorption for the GI and therefore increased urinary excretion of oxalic acid.[2,3,10] Hyperoxaluira increases the risk of calcium oxalate stones more than hypercalciuira, because you need less oxalic acid to form insoluble calcium oxalate.[2,3,9,10] High animal protein

diets increase the risk of calcium oxalate crystals due to increased urinary calcium excretion and decreased urinary citric acid excretion; citric acid chelates calcium so decreasing citric acid further increases calcium concentration in the urine.[2,9,10,14,15,17] Additionally, high animal protein diets may promote hypercalciuria by increasing glomerular filtration rate.[2, 9,10] Hyperadrenocorticism increases the incidence of calcium-containing stones by 10 times possibly due to decreased calcium reabsorption due to increased glomerular filtration rate in response to excess steroids.[2,9,10]

DIFFERENTIAL DIAGNOSES

Differential diagnoses for urolithiasis in general are similar for any cause of urinary blockage or urinary clinical signs and they include the following: urinary tract infection, cystitis, ectopic ureters, nephrolithiasis, pyelonephritis, and neoplasia. More specifically, if the stones are radiopaque on abdominal radiographs, they are either composed of calcium oxalate or contain phosphate, which includes struvite and hydroxyapatite stones.[3,8,9]

DIAGNOSTIC APPROACH/CONSIDERATIONS

A minimum database consisting of complete blood count, blood chemistry panel, and urinalysis should be performed prior to any advanced diagnostic procedures. The most definitive diagnostic tool for diagnosing uroliths is through imaging.[2,3,15] Calcium oxalate stones are radiopaque, and therefore can be detected on abdominal radiographs – they are often small and irregularly-shaped with a rough surface texture.[2,4,8,9] Ultrasound or double-contrast cystography are useful to detect radiolucent stones that will not show up on abdominal radiographs due to their small size or lack of radiodense composition.[2,8,9] In the absence of crystaluria, urine pH and specific gravity can help assess how likely uroliths are in the environment of a urine

sample.[2,3,13,15] That is, a high urine specific gravity gives evidence of highly concentrated urine and therefore more concentrated levels of urolithic precursors, and certain stones grow more readily in different pH ranges (purine, cystine, and calcium oxalate stones form in urine with a pH of less than 7.0, and struvite stones form in urine with a pH greater than 7.0).[2,3,13,15] It is important to rule out urinary tract infections via a urinalysis and urine culture due to the fact that certain uroliths are more likely in the presence of a urinary tract infections (struvite) and that uroliths can predispose patients to urinary tract infections.[3,5,8,10,15]

TREATMENT AND MANAGEMENT OPTIONS

If the stones are too large to pass through the urethra, they must be removed via cystotomy (surgical or laparoscopic) since calcium oxalate stones cannot be dissolved.[2,3,7,8,9,10,13,15,20] If the stones are small, they can be removed without surgery through a transurethral catheter or by aiding their passing via voiding urohydropropulsion or via cystoscopy.[7,19,21] Another non-surgical treatment option is traditional lithotripsy, although this is less effective for uroliths than it is for nephroliths due to difficulty of positioning and the fact that multiple lithotripsy treatments might be required to adequately reduce large uroliths.[2,21] However, laser lithotripsy can be utilized for a few large stones in the bladder or small stones obstructing the urethra as a non-surgical method to relieve urethral obstruction.[6,21]

Once the stones are removed, management is key to prevent recurrence of calcium oxalate stones. Increased water intake should be encouraged via canned diets, and a diet that does not over-acidify the urine and contains moderate to low levels of protein should be utilized – some diets that fit this criteria include the following: Hill's c/d Multicare, Hill's g/d, Hill's u/d, and Hill's Science Diet Mature Adult Gourmet Beef or Turkey.[15] Diet alone will most likely not prevent calcium oxalate stone formation completely, but it can slow the progression of

recurrence of stones.[4,15] Therefore, it is very important to serially monitor these patients for recurrence so early non-surgical intervention techniques can be utilized.[4,15] Urinalyses should be performed every 3 to 6 months to make sure that the urine pH is between 6.5 and 8.0 and the urine specific gravity is at 1.020 or lower.[2,3,915] Abdominal radiographs should also be performed every 6 to 12 months.[2,3,915] If the urine pH is consistently below 6.5, potassium citrate can be added to the therapeutic plan at a dose of 75 mg/kg every 12-24 hours.[2,9,15] In dogs that have recurrent urolithiasis without hypercalcemia, hydrochlorothiazide can be considered dosed at 2 mg/kg every 12 hours.[2,9,15]

EXPECTED OUTCOME AND PROGNOSIS

Prognosis for this condition is excellent if they are properly managed through diet and serial monitoring through urinalyses and abdominal radiographs. With this management, stone formation is less likely and it's more likely that small stones will be caught early while they're small enough to be passed via non-surgical means.[3,7,19,21]

CONCLUSION

In conclusion, calcium oxalate stones account for about 40% of urolithiasis in both the canine and feline patient.[2,3,10,14,15] Younger toy and small breed neutered male dogs are predisposed to this type of stone, with it being most common in Bichon Frise, Lhasa Apsos, miniature Schnauzers, Pomeranians, Shi Tzus, and Yorkshire terriers.[1,3,8,9,10,13] This type of stone cannot be medically managed, so they must be removed initially and then the patient needs to be managed with an appropriate diet and serial monitoring of urinalysis and abdominal radiographs.[2,3,8,9,10,13,15] Once the stones are removed and the patient is properly managed, calcium oxalate stones have a good to excellent prognosis.

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