Urolithiasis, Calcium Oxalate (Canine)

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Disease Description

Definition

Calcium oxalate uroliths are typically composed of 100% calcium oxalate, although a few contain some amounts of calcium phosphate or carbonate. Calcium oxalate can be found in two crystalline forms, calcium oxalate monohydrate and calcium oxalate dihydrate.[1,2]

Prevalence

Along with struvite uroliths, calcium oxalate uroliths are one of the most common uroliths in dogs and cats.[3,4] Calcium oxalate uroliths accounted for 42% of 25,499 canine uroliths in one study and 44.5% of 468 canine uroliths in another study.[3,5] The percentage of calcium oxalate submissions from dogs to the Minnesota Urolith Center increased from 5% in 1981 to 41% in 2007.[6] The proportion of calcium oxalate urolith submissions rose in 2009 and 2010 compared to 1999 and 2000 on every continent except Europe.[6,18-21]

Calcium oxalate uroliths accounted for 51.4% of 855 feline admissions in one report.[5] The prevalence of calcium oxalate uroliths in cats has increased dramatically since the 1980's. Between 1981-2004, the frequency of feline calcium oxalate urolith submissions to the Minnesota Urolith Center increased more than 50-fold[6,7]

Etiology and Pathophysiology

Numerous risk factors have been identified for development of calcium oxalate urolithiasis. Patients that form calcium oxalate uroliths have increased urinary excretion of calcium (hypercalciuria) and oxalate (hyperoxaluria).[8,9,22]

Hypercalciuria can occur through a variety of mechanisms, including serum hypercalcemia; increased intestinal absorption of calcium; impaired renal tubular reabsorption of calcium; and increased bone demineralization.[1] Hypercalciuria has been associated with a variety of disorders in dogs and cats, including hyperadrenocorticism, exogenous glucocorticoid usage, primary or secondary hyperparathyroidism, neoplasia, or other disorders that lead to hypercalcemia.[1,2] One study of canine breeds predisposed to calcium oxalate formation (e.g. miniature schnauzer, bichon frise, shih tzu) showed that urine calcium:creatinine ratios were higher in dogs with a history of uroliths compared to controls.[8]

Hyperoxaluria is also thought to be involved in formation of calcium oxalate crystals and uroliths. Urinary oxalate is derived from endogenous production in the liver from metabolism of ascorbic acid, glyoxylate, and glycine. Dietary protein sources also contribute amino acids that are metabolized to oxalate.[1] Excessive intestinal absorption of oxalate from a deficiency of *Oxalobacter formigenes* may be involved in hyperoxaluria. *O. formigenes* is part of the normal enteric flora and it degrades oxalate in the intestinal tract.[1,7,9] Two studies showed that the fecal microbiota of dogs with calcium oxalate uroliths differed from dogs without calcium oxalate uroliths.[10,24] However, the role of hyperoxaluria was disputed in another study that showed (in three canine breeds) the urinary oxalate:creatinine ratio was no different in dogs with calcium oxalate uroliths compared to breed-matched controls.[8] Cats with calcium oxalate uroliths excrete similar amounts of oxalate in their urine as cats without uroliths.[7]

Dietary factors may also lead to increased formation of calcium oxalate uroliths. In several studies, patients with calcium oxalate uroliths were fed diets lower in protein, fat, sodium, calcium, phosphorus, moisture, magnesium, potassium, and chloride compared to those without calcium oxalate uroliths.[11,12] Diets higher in carbohydrates were associated with an increased risk of calcium oxalate urolith formation in dogs.[11,12] Obesity has been shown to be a risk factor in dogs and cats.[1,23]

Diagnosis

History/Physical Examination Findings: Some patients with calcium oxalate urolithiasis are asymptomatic. Lower urinary tract signs are the most common signs and may include hematuria, dysuria, stranguria, pollakiuria, and urinary incontinence.[1,2] Other abnormalities may be reflect other underlying disease (e.g. hypercalcemia, hyperadrenocorticism). More severe signs (e.g. vomiting, depression, abdominal pain, bradycardia, cardiac arrhythmias) may occur in animals with urethral obstruction. Clinical signs of kidney disease may be present in patients with nephro- or ureterolithiasis.[2,9]

Serum Biochemistry Panel: Approximately 25-30% of cats with calcium oxalate uroliths are hypercalcemic.[1,9] Idiopathic hypercalcemia is the most common cause. Approximately 4% of dogs with calcium oxalate uroliths are hypercalcemic, with primary hyperparathyroidism being the most common cause.[9] Renal azotemia may be present in with nephro- or ureterolithiasis.[9]

Urinalysis: Abnormalities may include hematuria, pyuria, proteinuria, and bacteriuria.[1,7] Calcium oxalate crystals are present in the urine of approximately 50% of animals with calcium oxalate uroliths.[9] Calcium oxalate crystals and uroliths tend to form in acidic to neutral urine. Calcium oxalate crystals typically appear as square envelopes in the dihydrate crystal form (**Figure 1**) or as dumbbell or picket-fence shapes (**Figures 2A, B**) in the monohydrate form.[2]

Urine Culture: A urine culture is performed to determine if a secondary urinary tract infection is present.[2,13]

Calcium Oxalate Risk Index: The calcium oxalate risk index (CORI) can be calculated by titrating urine with sodium oxalate solution and observing it at 585 nm until precipitation occurs. CORI is calculated by dividing urine calcium concentration by the amount of oxalate added at the point of precipitation. CORI is influenced by allowing urine to cool to room temperature. CORI evaluation may be helpful in predicting which patients are more likely to form calcium oxalate uroliths.[14]

Abdominal Imaging: Calcium oxalate uroliths are moderately to markedly radiopaque (**Figures 3A, B**). Single or multiple stones may be present.[1,2] Calcium oxalate uroliths are most commonly found in the urinary bladder and urethra rather than the ureter and renal pelvis. However, calcium oxalate uroliths are the most common form of nephroliths and ureteroliths.[2,9] Small uroliths may not be detected on survey radiographs. The failure rate for detection of uroliths on survey radiographs ranges from 2-27%.[2] Abdominal ultrasonography or contrast radiography may be helpful in visualizing some uroliths.

Cystoscopy: Cystoscopy can be used to visualize uroliths in the lower urinary tract.[1]

Urolith Mineral Analysis: Quantitative mineral analysis is used to confirm the composition of surgically removed uroliths (**Figures 4A, B**). Uroliths should not be placed in formalin when shipping them to laboratories since this can lead to misdiagnosis of mineral composition.[25]

Disease Description in This Species

Signalment

Calcium oxalate uroliths are one of the most common uroliths found in dogs, accounting for 40-50% of canine uroliths.[3,4,5,9,18-21] Calcium oxalate uroliths tend to occur more often in male than female dogs.[4,23] In one study, 68-71% of calcium oxalate uroliths submitted to two urolith centers were removed from male dogs.[1,3] Predisposed breeds include the miniature schnauzer, Lhasa apso, Yorkshire terrier, bichon frise, pomeranian, shih tzu, cairn terrier, Maltese, miniature poodle, and chihuahua.[1,3,11,15,19] The mean age in dogs is 7-8.5 years.[11,15,23] Interestingly, dogs living in high income groups in Canada had a higher proportion of calcium oxalate urolithiasis than dogs living in low income families.[23]

Clinical Signs

Some patients with calcium oxalate urolithiasis are asymptomatic. Lower urinary tract signs are the most common and may include hematuria, dysuria, stranguria, pollakiuria, and urinary incontinence.[1,2] Other abnormalities may reflect other underlying diseases (e.g. hypercalcemia, hyperadrenocorticism). More severe signs (e.g. vomiting, depression, abdominal pain, bradycardia, cardiac arrhythmias) may be present with urethral obstruction. Clinical signs of kidney disease may occur with nephroureterolithiasis.[2,9]

Etiology

Diet
Glucocorticoids
Hyperadrenocorticism
Hypercalcemia
Hypercalciuria
Hyperoxaluria
Hyperparathyroidism, primary
Idiopathic, unknown

Breed Predilection

Bichon frise
Cairn terrier
Chihuahua
Lhasa apso
Maltese
Miniature poodle
Miniature schnauzer
Pomeranian
Poodle
Shih tzu
Yorkshire terrier

Sex Predilection

Male

Age Predilection

Mature, middle-aged

Clinical Findings

Abdominal pain
AFEBRILE
Anorexia, hyporexia
Bradycardia
Dehydration
Depression, lethargy
DYSURIA, STRANGURIA
FEVER
Hematuria
HEMORRHAGE
HYPERSALIVATION
Kidney pain
Nausea
PAIN
Pollakiuria
Polydipsia
Polyuria
TACHYCARDIA
TENESMUS
Urinary bladder distension

Urinary bladder painful
Urinary bladder thickened
Urinary incontinence
Urine discolored, cloudy
Urine odor unusual or foul smelling
UROLITHIASIS
VOMITING
Weight loss
ZZZ INDEX ZZZ

Diagnostic Procedures

Diagnostic Procedures:	Diagnostic Results:
Urinalysis	Bacteriuria, urine bacteria increased Calcium oxalate crystals CRYSTALLURIA, URINE CRYSTALS INCREASED Proteinuria, albuminuria Pyuria, increased white blood cells Red blood cells present in urine Urine acid, pH decreased
Radiography of abdomen	Renal or bladder lithiasis Ureteral urolith Urethral urolith
Electrocardiography (ECG)	ARRHYTHMIA, CARDIAC IRREGULARITY ELECTROCARDIOGRAM ABNORMAL Sinus tachycardia Tachycardia, ventricular
Serum biochemistries	Azotemia/uremia Hypercalcemia
Culture of urine	Aerobic culture may be positive for pathogen
Radiography, contrast procedure	Urethral obstruction
Urolith analysis	Oxalate uroliths

Images

Click on each image to see a larger view

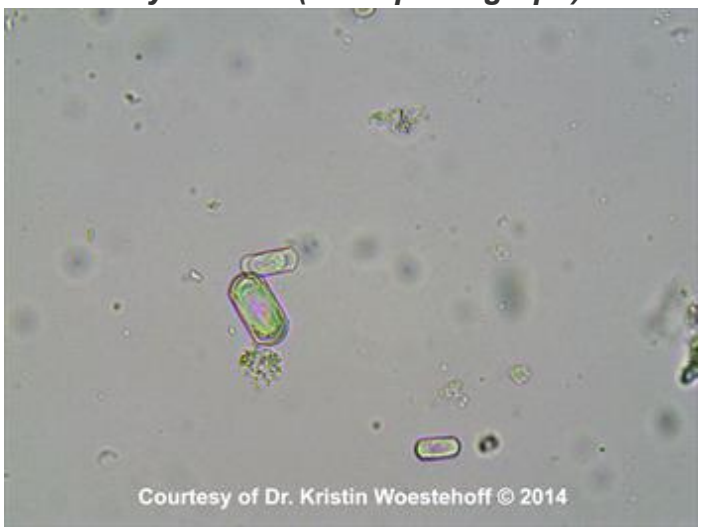
Figure 1. Calcium oxalates - rhomboid, dihydrate

Urine, 10x

Figure 2A. Monohydrate oxalate crystal

Urine, 40x. Courtesy Dr. Sally Lester.

Figure 2B. Calcium oxalate monohydrate crystalluria (microphotograph)



"Daughter cells" are visible within these calcium oxalate monohydrate crystals seen in a dog's urine sediment.
Click here to see board discussion

Figure 3A. Ca oxalate stones & prostatomegaly

Click here to see board discussion

Figure 3B. Ca oxalate stones in prostate

Click here to see board discussion

Figure 4A. Calcium oxalate stones

Click here to see board discussion

Figure 4B. Calcium oxalate dihydrate calculi

Treatment / Management

SPECIFIC THERAPY

Calcium oxalate uroliths are not amendable to medical dissolution; therefore, they must be physically removed.[1,2,9]

Voiding urohydropropulsion can be done to remove small uroliths by inducing voiding and encouraging the uroliths to pass through the urethra with voided urine. The urinary bladder is distended with sterile saline either via cystoscopy or urethral catheterization. The patient is positioned vertically so the spine is 25°caudal to a line perpendicular to the effects of gravity. The bladder is agitated to allow the uroliths to fall into the bladder trigone, and then the bladder is expressed to promote voidance of the uroliths. The process is repeated until no uroliths are noted on imaging (i.e. cystoscopy, ultrasonography, radiography).[1,16] Anesthesia or sedation is unnecessary but is helpful in performing the procedure. Uroliths <5 mm can typically be removed through voiding urohydropropulsion from dogs >8 kg in body weight.[16]

Cystoscopic retrieval of small uroliths or urolith fragments can also be performed. A urolith basket is used to snare uroliths that are smaller than the diameter of a distended urethra.[9,16]

Laser lithotripsy involves placing a Holmium:YAG laser in direct contact with uroliths (via cystoscopy) for fragmentation.[26-28] The fragmented pieces are then removed via cystoscopy or voiding urohydropropulsion. Laser lithotripsy had a reported success rate of 83-100% for female dogs and 68-81% for male dogs.[1,16] Some male dogs and cats are too small for laser lithotripsy to be successful. Furthermore, some large uroliths may be difficult to fragment into pieces small enough to allow for removal.[16]

Extracorporeal shock wave lithotripsy (ESWL) involves fragmentation of uroliths by using shock waves generated outside the body.[29] A wet lithotripter requires the patient to be partially submerged in a water bath. A dry lithotripter generates shock waves through a water-filled cushion.[16] ESWL can be used to fragment cystic calculi but it is best suited for treatment of immobile uroliths, such as nephroliths or ureteroliths.[1] Cystic calculi tend to move out of the focal spot of the lithotripter.

Surgical removal via a cystotomy (e.g. via abdominal incision, laparoscopic-assisted), ureterotomy, or urethrotomy may also be considered.[9,16] Complications with cystotomy is uncommon but ureterotomy and urethrotomy have potentially have more complications (e.g. stricture formation, hemorrhage, urinary leakage, surgical dehiscence).

SUPPORTIVE THERAPY

Thiazide diuretics decrease urinary calcium excretion in dogs with calcium oxalate urolithiasis and may be beneficial in reducing the risk of calcium oxalate urolithiasis.[1,9] Either hydrochlorothiazide (2 mg/kg PO q 12 hrs) or chlorothiazide (20-40 mg/kg PO q 12 hrs) may be considered.[9]

Treatment of underlying conditions that are contributing to increased risk of calcium oxalate urolithiasis is warranted.

MONITORING and PROGNOSIS

Calcium oxalate uroliths have a high rate of recurrence.[1,9,11] Repeated urinalyses, with sediment examination, are done to ensure the desired pH of 7.5 and urine specific gravity of <1.020 are present. Abdominal radiographs are evaluated q 6 months as part of routine monitoring for recurrence.[1]

Preventive Measures

Calcium oxalate uroliths have a high rate of recurrence. Recurrence rates up to 50% within 3 years have been reported.[1] Miniature schnauzers have three times the risk of recurrence compared to other breeds.[11] Preventive therapy is recommended because of this high rate of recurrence.

Dietary therapy is one of the cornerstones of preventive therapy for calcium oxalate uroliths. The ideal composition of a diet for calcium oxalate prevention is not completely known.[1] Dietary factors associated with a decreased risk of calcium oxalate urolithiasis include increased dietary water, protein, calcium, phosphorus, magnesium, sodium, potassium, and chloride.[1,11,17,30] Increased dietary carbohydrates, phosphorus restriction, and acidifying diets are associated with an increased risk of calcium oxalate urolithiasis.[1,11] Diets recommended to reduce the risk of calcium oxalate

urolith formation include Royal Canin S/O Lower Urinary Tract Support Diet (Royal Canin USA), Hill's Prescription Diet w/d and u/d (Hill's Pet Nutrition).[1,9,11] Increasing water intake is an important component of prevention.[1]

Potassium citrate can help prevent calcium oxalate uroliths. Citrate forms a soluble salt with calcium; therefore, inhibiting calcium oxalate crystal formation. It also helps to alkalinize the urine. Starting doses of 50-75 mg/kg PO q 12 hours are titrated to achieve a urine pH of approximately 7.5.[1,9]

Special Considerations

Other Resources

Recent VIN Message Board discussions on dietary considerations for calcium oxalate urolithiasis
Recent VIN Message Board discussions on calcium oxalate urolith prevention
University of Minnesota Urolith Center's recommendations on calcium oxalate urolithiasis
Client Handout on oxalate bladder stones

Proceedings articles that discuss calcium oxalate urolithiasis
Medical FAQ on Urinary Incontinence Localization
2016 ACVIM Small Animal Consensus Recommendations on the Treatment and Prevention of Uroliths in Dogs and Cats

VIN Mentor Visual Procedures Manual Video on Cystotomy
For more images see the Cystic calculi - xrays - Dog slideshow in the Image Library

Differential Diagnosis

Cystitis, bacterial
Other types of uroliths: urate, calcium phosphate, cystine, struvite, silica, xanthine
Urethritis
Urinary bladder & urethral neoplasia
Urinary tract trauma

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