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Uroliths form because the amount of the crystal involved exceeds its solubility in urine. Characteristics of urine that affect solubility include concentration of the crystal, urine pH, presence of crystallization inhibitors and promoters, and the proteinaceous matrix. The major questions related to urolithiasis are what type of urolith is present and where uroliths are located.

Clinical Signs

Most nephroliths produce no clinical signs. Occasionally nephroliths cause hematuria or vague malaise. If bilateral, nephroliths can result in chronic renal failure. Most ureteroliths are also asymptomatic. Possible signs are abdominal pain, vomiting, or hematuria. If a ureterolith causes prolonged partial obstruction, hydroureter and hydronephrosis result. Some cystoliths are asymptomatic. Other cystoliths produce dysuria and/or hematuria. Urethral calculi are the predominant cause of urethral obstruction with the most prominent sign being dysuria.

Diagnosis

Uroliths are diagnosed by palpation or by radiography/ultrasonography. The type of urolith is determined by quantitative stone analysis. An educated guess as to the type of urolith can be made by considering signalment, history of previous stone formation, urinalysis (especially urine pH), urine culture, serum calcium concentration, and radiodensity.

Therapy

If calculi are confined to the bladder and are smaller than the diameter of the urethra, they can be removed by urohydropulsion.

Under saturation of urine with the mineral components of the urolith will prompt dissolution. Because uroliths must be bathed in urine for dissolution, dissolution is most successful for cystoliths, requiring approximately 2-4 months. Nephroliths may be dissolved if renal function is adequate in the affected kidney as determined by excretory urography. A longer time is required to dissolve nephroliths (mean for struvite nephroliths is 6 months). Animals that are being treated to dissolve calculi must be reevaluated during therapy with CBC, biochemical profile, urinalysis, urine culture, and radiography. The evaluations are performed at 4 week intervals. Therapy and these evaluations are continued at least 4 weeks beyond the last radiographic evidence of uroliths.

Uroliths tend to recur. A general recommendation to prevent recurrence is to increase water intake.

Struvite (Magnesium Ammonium Phosphate) Uroliths

Pathophysiology: Struvite crystals are found in urinalyses from normal dogs because dogs excrete magnesium, ammonium, and phosphate. The most common etiology of urolith formation is urinary tract infection with bacteria that produce urease (staphylococci, *Proteus* spp) causing urine alkalinization. Female dogs have a higher incidence than males. Struvite uroliths can affect any age animal. Sterile struvite uroliths occur occasionally. The stone should be cultured when urine is sterile.

Dissolution of Infection Induced Struvite Uroliths: Dissolution requires control of infection with an appropriate antimicrobial and reduction of urine concentrations of ammonium, magnesium, and phosphate usually by diet. Canine s/d (Hills Co., Topeka, KS) is formulated for struvite urolith dissolution.

Prevention of Recurrence of Infection Associated Struvite Uroliths: It is most important to monitor for infection. A urinalysis and urine culture should be performed one week after discontinuing antibiotics and every month thereafter for 3 months. The owner should monitor urine pH several times a week on urine collected in the morning prior to feeding. If the owner notes urine pH > 7.0 for more than 3 days in a row, the owner should return the pet for urinalysis and urine culture.

Calcium Oxalate Uroliths

Pathophysiology: Hypercalciuria is thought to be the important factor in formation. Most affected dogs are normocalcemic. However, hypercalciuria may be secondary to hypercalcemia.

Medical Therapy: Calcium oxalate uroliths cannot be dissolved. Calcium oxalate cystoliths may be able to be removed by urohydropulsion or cystoscopy.

Prevention of Recurrence: If the dog is hypercalcemic, it is most important to diagnose and treat the cause.
Acidifying diets should not be used. Diets designed for prevention of calcium oxalate stones, diets designed for renal failure, or high fiber diets can be fed. Hydrochlorothiazide (2-4 mg/kg every 12 hours) has been shown to reduce calciuria in dogs, probably by increasing calcium reabsorption. Thiazide diuretics should never be used in animals which are hypercalcemic and serum calcium should be monitored weekly for the first few weeks. Citrates chelate with calcium to form soluble salts. Citrates also alkalinize urine. Potassium citrate is usually used. A urine pH of 7.0 suggests that adequate urine citrate concentrations have been achieved. Salt should not be used to stimulate thirst, since salt increases urine calcium excretion.

Once a preventative regimen is established, the dog should be reevaluated every 3-6 months with a urinalysis and survey abdominal radiographs to detect calculi while they are small and removable by urohydropulsion.

**Urate Uroliths**

Pathophysiology: 90% of urate calculi are ammonium urate. Dalmatians (and probably bulldogs) are genetically predisposed to urate uroliths. Urate uroliths are most commonly detected in males and are most frequent between 3-6 years. The ability of the liver of Dalmatians to oxidize uric acid to allantoin is intermediate between other breeds and humans. This leads to a urine uric acid concentration that is approximately 10 times the concentration in other breeds and results in urate crystals in urine of normal Dalmatians. Only a small percentage of Dalmatians develop calculi, indicating that other factors are important. The major cause of urate uroliths in other breeds is portovascular shunt or, more rarely, hepatic dysfunction.

Dissolution of Urate Uroliths Unassociated with Portovascular Shunts: Allopurinol, alkalinizing the urine, modifying the diet, and increasing water intake are used. Allopurinol decreases the formation of urate by inhibiting xanthine oxidase which normally converts xanthine to uric acid. The dosage is 30 mg/kg/day divided into 2 or 3 doses. Urine alkalization is usually accomplished with sodium bicarbonate or by diet. The dosage of sodium bicarbonate should maintain a urine pH of 7.0-7.5. The goal of dietary modification is to reduce uric acid intake which is primarily contained in purine containing glandular organs (e.g. kidney, liver), to promote urine alkalinization, and to promote diuresis. Diets designed for renal failure can be used.

Treatment of Urate Uroliths Associated with Portovascular Shunts: Surgical correction of the shunt should be performed.

Prevention of Recurrence: Urine alkalinization is recommended. Canine u/d can be fed. Dilated cardiomyopathy has been reported in Dalmatians eating u/d for longer than 6 months (an average of 33 months). Alternatively, allopurinol can be used at 10-20mg/kg/day divided into two doses. Appropriate therapy can be determined by monitoring 24 hour urine urate excretion with a goal of 275-325 mg of urate excreted in 24 hours. If 24 hour collections cannot be performed, urine urate/urine creatinine ratios can be used. A reduction in urine urate/urine creatinine ratio from 0.5-0.8 pre-treatment to 0.25 to 0.3 post-treatment is recommended. Recurrence was reported in 30% of dogs despite preventative measures. Dogs with urate urolithiasis should be examined every 3 months with a urinalysis and radiographs and/or ultrason sound to detect uroliths when small and removable by urohydropulsion.

**Cystine Uroliths**

Pathophysiology: Affected dogs have a renal reabsorptive defect for cystine. Predisposed sex/breeds are male dachshunds and English bulldogs.

Dissolution: Diet (canine u/d) and administration of thiola are used. The dosage of N-(2-mercaptpropionyl)-glycine (MPG; thiola) is 15 mg/kg per os every 12 hours. One reported side effect is nonpruritic vesicular skin lesions. Dosage reduction can lead to resolution of skin lesions. Another is Coomb’s positive regenerative anemia.

Prevention: MPG is used in sufficient quantity to keep the urine cystine concentration below 200 mg/L. If urine cystine cannot be measured, MPG is given at 30 mg/kg/day. Sodium bicarbonate or potassium citrate can be used to alkalinize the urine. Urine pH should be maintained at about 7.5. Alternatively, a low protein diet that promotes alkaline urine (renal failure diet) can be fed.