Management of Calcium Oxalate Uroliths in Cats and Dogs

Management of CaOx uroliths for dogs and cats is very similar and therefore this section will focus on the management of these stones for both species. There is no dissolution protocol for CaOx uroliths, so removal and quantitative mineral analysis of the stones should be performed if they are growing or causing clinical disease. Less invasive techniques are now available for stone removal including voiding urohydropropulsion, Holmium: YAG laser lithotripsy, basket retrieval through the cystoscope, and laparoscopy with or without the use of the laser. After the CaOx (or any urolith) has been removed, it should be submitted for a complete quantitative crystallographic analysis.

Animal related factors should be addressed initially to ascertain that no intrinsic predisposing problems are present. If serum calcium concentration is elevated, a search should be initiated for underlying causes such as primary hyperparathyroidism, neoplastic processes and idiopathic hypercalcemia in cats.\(^1\) Research in rats suggests that elevated lipids may play a role in CaOx urolith formation\(^2\) therefore screening animals to evaluate fasted serum triglycerides concentration may be warranted, particularly in breeds prone to hyperlipidemia such as the miniature schnauzer. A baseline urinalysis and urine culture should be performed with special attention given to the evaluation of the urine specific gravity and urine sediment. Urine samples should be analyzed within 60 minutes of collection to minimize temperature- and time-dependent effects on in vitro crystal formation. Presence of crystals observed in stored samples should be reevaluated by analyzing a fresh sample.\(^3\)

A diet high in moisture is recommended to decrease the urine concentration of mineral precursors. This can be achieved most easily by feeding a canned diet. Alternatively, water can be added to the dry kibble to achieve higher moisture content if the pet will not consume a canned diet, although 85% moisture is difficult to attain using this method. Gradual introduction of the high moisture diet is important to prevent initial diarrhea. In one study\(^4\) feeding a high moisture diet significantly reduced the relative supersaturation of CaOx in healthy miniature schnauzers. Other studies suggest that dogs that consumed diets highest in moisture were six times less likely to form CaOx uroliths as compared with control dogs.\(^5\) If the animal does not accept and tolerate an increase in dietary moisture content, attempts can be made to increase voluntary water intake by using drinking fountains, and by adding flavored juices (ham, tuna) or ice cubes to the pet’s drinking water. As an initial guideline, a specific gravity of <1.020 for dogs and <1.025 for cats seems reasonable. Urine specific gravity should be monitored at periodic reevaluation until the adequacy of the current strategy is assessed. Urine specific gravity should be evaluated from home samples to gain a better understanding of owner compliance and water intake in the home environment.

The addition of sodium chloride (salt) to the diet is another method of increasing voluntary water intake. Current recommendations for appropriate salt intake in animals with prior CaOx uroliths are controversial, but sodium chloride supplementation has been reported to increase urinary calcium excretion and increase the risk for CaOx uroliths in people.\(^6\) Caution should be practiced if using dietary salt supplementation to manage lower urinary tract signs, particularly in animals with reduced renal function, cardiac disease or hypertension. After utilizing strategies to increase the moisture content of the diet, if the urine is still too concentrated and/or urine sediment findings...
are not ideal, one can try the addition of salt to the diet to increase the urine volume produced daily. A series of studies using healthy dogs showed no effect of added dietary salt on urine calcium or oxalate concentrations. Studies have also shown that increasing the dietary sodium content resulted in a decrease in the CaOx relative supersaturation (RSS) in both miniature schnauzers (a breed predisposed to CaOx urolithiasis) and Labrador retrievers (a breed thought to be at lower risk of CaOx urolithiasis). No increases in urinary calcium concentrations were found in either breed when dietary sodium concentrations as high as 3 grams/1000kcals were fed. No adverse effects of added sodium chloride have been noted in short term studies in healthy cats.

Several commercially available canine and feline therapeutic diets are marketed as assisting to reduce the recurrence of CaOx uroliths. A diet that is restricted in both calcium and oxalate seems logical for animals prone to CaOx urolithiasis, but no evidence based studies in dogs or cats with naturally occurring disease are available to support or refute this recommendation. Moreover, higher intake of dietary calcium appears to decrease the risk for symptomatic kidney stones in humans. Stevenson, et al, evaluated the effects of dietary calcium and oxalate content when fed to stone forming (Miniature Schnauzers) and normal dogs. The lowest level of dietary calcium and oxalate resulted in the lowest CaOx RSS. The high calcium, low oxalate diet resulted in the highest CaOx RSS, a low calcium diet with increased dietary oxalate also tended to increase CaOx RSS although the results were highly variable. Urinary oxalate increased, although inconsistently, with increased dietary oxalic acid only when dietary calcium was low. In the intestine, non-absorbed dietary calcium is available to form complexes with phosphate, citrate, sulfate and oxalate, in turn preventing their absorption. Thus, the timing of calcium intake with intake of other nutrients may also influence stone formation, and this effect may be different between animals with CaOx urolithiasis and healthy animals. Reducing the dietary content of only one of the CaOx precursors could potentially increase the intestinal absorption and urinary excretion of the other. As with excess calcium, foods rich in oxalate or oxalate precursors should also be avoided. For a list of the oxalate content of foods, please see the Oxalosis and Hyperoxaluria Foundation website: http://www.ohf.org/diet.html.

Another nutrient to consider in the dietary management of CaOx urolithiasis is magnesium. Urinary magnesium, as along with urinary citrates and phosphates, are thought to act as inhibitors of CaOx urolith formation and therefore should not be restricted in the diet. Dietary phosphorus should not be excessively restricted because reduced serum phosphorus could result in increased activation of vitamin D3 to calcitriol by 1-α-hydroxylase in the kidney under the action of PTH and result in increased intestinal absorption of calcium. Avoidance of Vitamin C supplementation is recommended in humans, because it is a metabolic precursor of oxalate. A moderate to slightly increased protein content may also be beneficial to prevent calcium oxalate urolith formation. Recent epidemiologic studies have suggested that increased protein intake may be protective, likely because of protein associated increases in urine volume. Protein restriction does not seem warranted as a dietary strategy for the management of canine or feline CaOx urolithiasis.

Lastly, as mentioned above dietary fats have been speculated to be involved in CaOx stone formation in rats and humans. Although the pathogenesis of CaOx stone formation in dogs and cats, may differ, it may be prudent to feed dogs with an elevated fasting serum triglyceride concentration and/or pancreatitis a low fat commercially available canned diet (or low fat home cooked diet) with water added to assist with reducing urine specific gravity.
If dietary manipulations are unsuccessful in preventing CaOx urolith recurrence alone, drug therapy may provide additional benefit. Administration of citrate as potassium citrate (Urocit-K®) could be helpful because urinary citrate may act as an inhibitor of calcium oxalate formation. Hypocitraturia may be a risk factor for calcium oxalate stone formation in humans because citrate can become chelated to calcium, forming a more soluble salt than CaOx in the urine. This additive appears beneficial only to humans where citrate excretion is low. The recommended dosage of potassium citrate is 100-150 mg/kg/day for both cats and dogs, but it is unclear if this dosage will actually increase urinary citrate in cats. Dogs generally excrete much less citrate than humans (only about 3% of filtered citrate is excreted by dogs as compared with 10-35% in humans) so what, if any, benefit citrate supplementation has on calcium oxalate formation in this species is, as yet, undetermined.

Hydrochlorothiazide is a diuretic which decreases urine calcium excretion in humans and has been recommended to prevent recurrence of CaOx urolithiasis in dogs. A study in dogs reported that hydrochlorothiazide significantly reduced urine calcium excretion. The hypocalciuric effect of this drug may be negated with augmentation of oral fluid intake. A recent abstract was also published evaluating the effects of hydrochlorothiazide and calcium excretion in healthy cats. The administration of hydrochlorothiazide is contraindicated in cats or dogs with hypercalcemia.

References: