

Management of common uroliths through diet

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In [part one of this feature](#) (VT45.34), the author considered dietary therapy for chronic kidney disease (CKD). Part two looks at dietary management of uroliths in cats and dogs.

Struvite uroliths (stones)

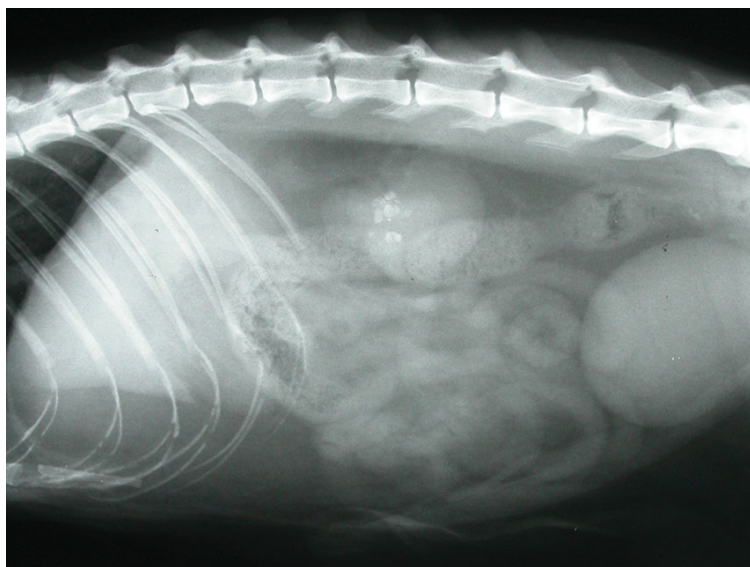


Figure 1. Radiograph of a cat showing uroliths, probably calcium oxalate in the kidney and ureter. Image: Daniëlle Gunn-Moore.

Struvite (magnesium ammonium phosphate) stones are one of the most common uroliths in dogs and cats. Struvite calculi, especially in the dog, are often associated with infection with urease-producing organisms (*Staphylococcus*, *Proteus* or *Ureaplasma*). Urease is an enzyme that breaks down urea, causing the release of ammonium and bicarbonate ions into the urine.

Supersaturation with ammonium ions promotes struvite formation, while the bicarbonate ions alkalinise the urine, also enhancing the likelihood of struvite crystals and stones. Since male dogs are less susceptible to urinary tract infections, struvite calculi are more common in female dogs. "Sterile" struvite uroliths may also occur in dogs and more often in cats despite the absence of infection.

Struvite crystals are normal in dogs and cats, but the presence of crystals with radiodense uroliths is an indicator of struvite uroliths. It is possible to have crystals of one type and a stone of another. Medical treatment for stone dissolution consists of antibiotic therapy when appropriate, plus dietary modification.

A calculolytic diet for struvite uroliths reduces urine levels of magnesium, ammonium and phosphate ions. These diets are usually acidifying and urinary acidifiers should not be added or supplemented. These diets should not be used in puppies or kittens as they are mineral restricted, and they should not be used in CKD patients or patients with idiopathic cystitis due to the acidification.

Therapeutic response may be monitored by urinalysis and culture, and abdominal radiographs. Surgery is indicated if a reduction in urolith size is not detected within several months, despite the clearance of infection and the production of acid urine.

When the stone does not decrease in size sufficiently, it is possible the owner cannot comply with the diet, the stone type was misjudged, or there is a stone of mixed minerals (such as partially calcium oxalate).

Calcium oxalate uroliths

Calcium oxalates are the second most common uroliths in dogs and may be the most common in cats. They are usually located within the bladder, but may also be found in the kidneys and ureters (**Figure 1**). Treatment of clinically significant uroliths is usually by surgery as there is no diet that will dissolve the stones. Sometimes, stones in the kidney are inactive, but stones blocking the ureter are a surgical emergency.

For these uroliths to form, the urine must be supersaturated with calcium. Factors that may affect supersaturation of calcium include hypercalcaemia (although serum calcium is often normal) and possibly a diet containing high protein, high calcium and low vitamin B6.

A decrease in the intestinal *Oxalobacter formigenes* bacteria, which metabolise oxalates, has been found in dogs that form oxalate-containing uroliths compared to those that do not (Gnanandarajah et al, 2012). Obesity is also a risk factor in humans and could possibly increase the risk in dogs and cats (Taylor et al, 2005). The solubility of calcium oxalate crystals is less directly influenced by urine pH within the physiologic range than struvite stones, but acidosis may increase the amount of calcium released from the bones to buffer the acid, resulting in hypercalciuria.

Preventing recurrence includes treatment of any underlying cause of hypercalcaemia, concurrent disease (such as hyperadrenocorticism), and increasing water intake to encourage formation of less concentrated urine. Decreased urine concentration can also be aided by adjusting the diet (and possibly with thiazide diuretics, if necessary). Diuresis (resulting in decreased urine

concentration) decreases the risk of calcium oxalate urolithiasis in dogs. Similarly, cats consuming canned (high moisture) diets have much less risk compared to cats on drier food. Formation of less concentrated urine lowers the risk of supersaturation.

Recommended dietary modifications include lower protein, with adequate, but not excessive, phosphorus, magnesium and potassium. Some researchers have found an increase in dietary sodium, resulting in a diuresis, decreases the relative supersaturation of calcium, even though it increases the total excretion.

Treatment in people includes use of potassium citrate and vitamin B6, but the efficacy of these in veterinary patients has not been proven. The use of probiotics, which increase oxalate metabolising bacteria in the intestines, is a potential future treatment.

Uric acid/urate uroliths

Urate calculi form because of increased excretion of urates or uric acid in the urine. Dalmatian dogs and bulldogs have a higher frequency of urate stone formation than other breeds. Idiopathic urate stones also may form in one to four-year-old cats, but the metabolic defect is unknown. Breeds that are predisposed to portosystemic shunts, such as Yorkshire terriers, may also form urate stones due to their liver disorder (**Figure 2**).

In normal animals, purines convert to hypoxanthine, which converts to xanthine, then into uric acid, which then converts to allantoin – a soluble end product excreted in urine. In Dalmatians uric acid is not converted to allantoin, resulting in urine that is oversaturated with uric acid. Dalmatians also have a lower percentage of renal tubular reabsorption than other breeds, resulting in increased urate excretion.

Not all Dalmatians form stones – male Dalmatians are reported as stone formers more often than females, but this may be because male dogs have more of a problem with urethral obstruction than females. The average age at which a Dalmatian is first found to have stones is 4.5 years and the risk of stone formation declines as the dog ages. Other risk factors include increased renal excretion of ammonium, low urine pH, and urinary tract infections with urease-producing bacteria – for example, *Staphylococcus*, *Proteus*, *Escherichia coli* and *Mycoplasma*, which may increase ammonium ions.

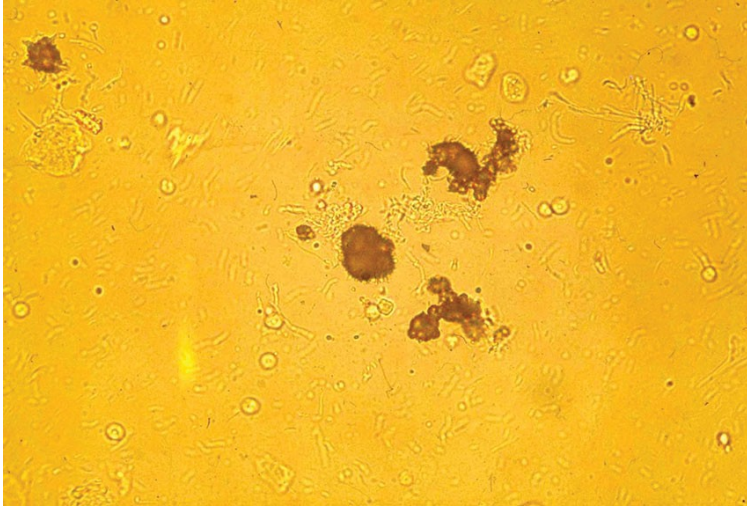


Figure 2. Urate crystals.

Dietary components may promote stone formation in predisposed dogs because dietary purines may be digested, absorbed and incorporated into the body's purine pool to be eventually excreted in the urine.

Detection of urate crystals or stones in breeds other than the Dalmatian or bulldog strongly suggests the presence of defective metabolism of uric acid due to profound hepatic dysfunction rather than an isolated familial metabolic defect. It is usually associated with either congenital portosystemic shunts or acquired liver failure.

Hepatic disease (such as portocaval shunts or cirrhosis) results in reduced conversion of uric acid to allantoin, which increases the rate of urinary ammonium excretion. Animals with portosystemic shunts may develop ammonium urate uroliths because of impaired metabolism of uric acid and ammonia.

In cases of urethral or ureteral obstruction or portosystemic shunts, surgery is required. Medical dissolution of ammonium urate stones can be attempted in other cases that do not have liver disease.

Dissolution involves using a low purine diet, a xanthine oxidase inhibitor (allopurinol), alkalinisation of urine, treatment of any urinary tract infection, and promoting increased quantity of less concentrated urine. Medical dissolution takes on average eight to 12 weeks and is successful in about one in three of affected dogs and partially successful in another third.

A calculolytic diet that is purine-restricted, non-acidifying and has no added sodium is recommended. It results in substantial reduction in urinary uric acid and ammonium excretion. English bulldogs have an associative risk of urate/cystine stones with dilated cardiomyopathy – feeding an “ultra-low” protein diet may precipitate cardiac disease, so a “renal failure” diet with a

low dose of allopurinol is preferred.

Allopurinol (used at 15mg/kg PO q12 hr) is a synthetic isomer of hypoxanthine that rapidly binds to, and inhibits, the action of xanthine oxidase, thereby decreasing production of uric acid by inhibiting conversion of hypoxanthine to xanthine and xanthine to uric acid. As prolonged administration of high doses of allopurinol may result in xanthine uroliths, it is used with dietary therapy, alongside the option of treating recurrent episodes of urate uroliths with dissolution protocols.

The urine should be alkalinised with oral sodium bicarbonate (25mg/kg q12 hr to 50mg/kg q12 hr) or potassium citrate (75mg/kg q12hr and adjust as needed) to maintain a urine pH of approximately seven, which will help reduce renal tubular production of ammonia. Sodium bicarbonate may be better at alkalinising the urine, but increases sodium intake and can combine with uric acid to form sodium urate.

Some diets (for example, many of those formulated for CKD) contain potassium citrate. Prevention of reoccurrence is similar to the protocol for medical dissolution. For cases caused by liver disease or portosystemic shunts, the underlying liver disease should be addressed.

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