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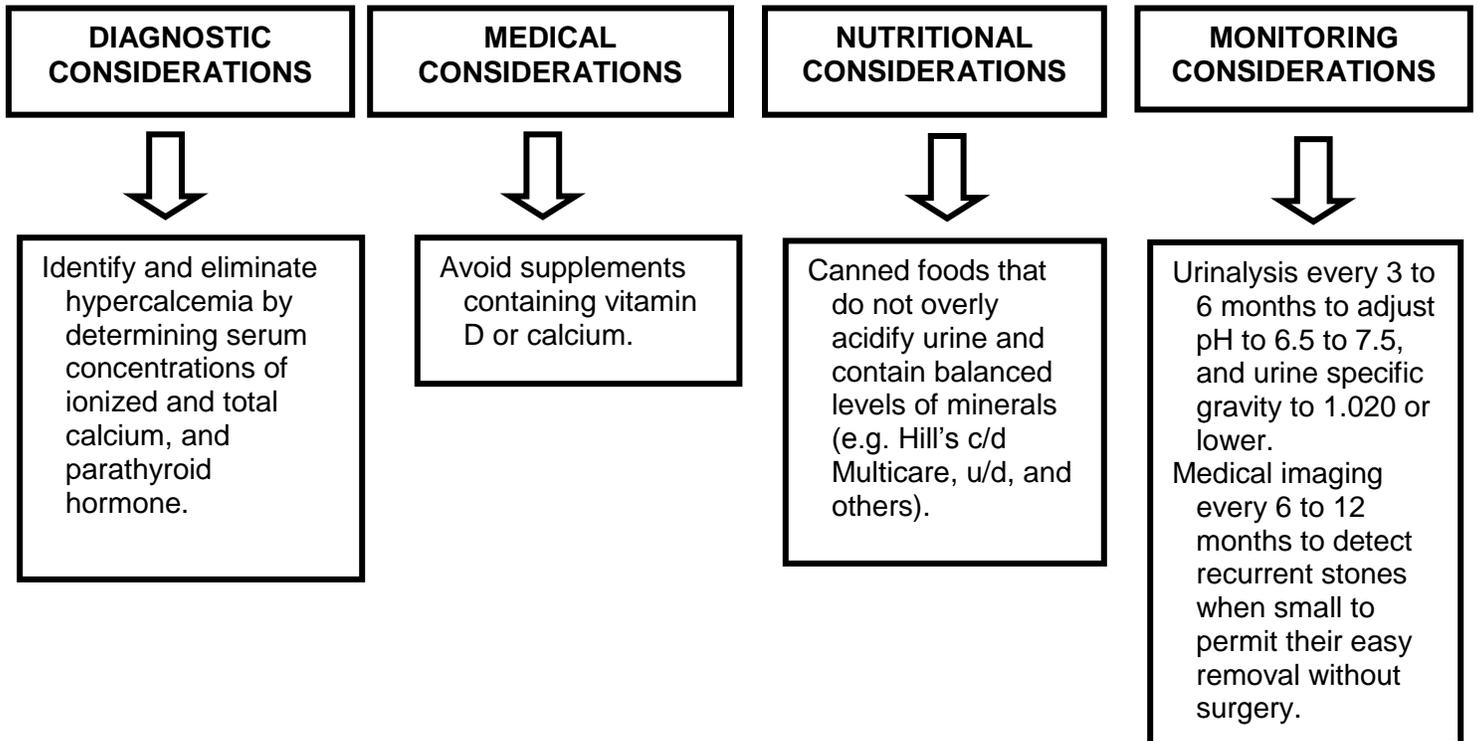
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## CANINE CALCIUM PHOSPHATE

Calcium phosphate uroliths (hydroxyapatite, brushite, whitlockite, and octacalcium phosphate) are uncommon in dogs. Common conditions associated with these minerals include hypercalcemia, hyperparathyroidism, hypervitaminosis D, and dystrophic and ectopic mineralization of vital tissues (blood clots, urothelium, etc.).

## PREVENTION



\*\* We advise reviewing manufacturer's literature regarding selected therapeutic foods to determine indications and contraindications. For pets with multiple health concerns, we suggest that the selection of diet should take into consideration all health needs of the pet.

**In depth recommendations and references are available on our website: [urolithcenter.org](http://urolithcenter.org) under the resources tab.**

## Minnesota Urolith Center

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**I will not use the knife, not even on sufferers from stone, but first will consult with individuals engaged in this work.**

**Modified from the Hippocratic Oath**

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### CANINE CALCIUM PHOSPHATE UROLITHS

The most common forms of calcium phosphate observed in canine uroliths are hydroxyapatite and carbonate-apatite. The name carbonate-apatite is derived from the fact that carbonate ion may displace phosphate ion in some uroliths. Less common forms of calcium phosphate include calcium hydrogen phosphate dihydrate (Brushite), tricalcium orthophosphate (Whitlockite), and octacalcium phosphate.

Calcium phosphate is commonly found as a minor component of struvite and calcium oxalate uroliths. Uroliths composed principally of calcium phosphate are uncommon in dogs, and are usually associated with metabolic disorders such as primary hyperparathyroidism, renal tubular acidosis, and excessive dietary calcium and phosphorus.

Surgery remains the most reliable way to remove active calcium phosphate uroliths from the urinary tract. However, we emphasize that surgery may be unnecessary for clinically inactive calcium phosphate uroliths. We have documented nephroliths composed of blood clots mineralized with calcium phosphate. Formation of highly concentrated urine in patients with gross hematuria may favor formation of blood clots. Mineralized blood clots may remain inactive for long periods, thus surgical removal is not always warranted. Small urocystoliths may be nonsurgically removed by voiding urohydropropulsion, or by aspiration through a urinary catheter.<sup>1</sup> Medical therapy of patients with recurrent calcium phosphate uroliths should then be directed at removing or minimizing risk factors that contribute to supersaturation of urine with calcium phosphate.

Patients with hypercalcemia and primary hyperparathyroidism usually require surgery. Parathyroidectomy may result in dissolution of uroliths and generally prevents their recurrence.

Several different medical protocols have been reported to be of value in humans with normocalcemic hypercalciuria. Ideally, the choice of therapy should be based on the cause of idiopathic hypercalciuria.

- a. There has been little clinical experience in the use of drugs in dogs and cats with calcium phosphate uroliths. However, medications which can enhance calcium excretion such as Glucocorticoids, furosemide, and those containing large quantities of sodium should be avoided if possible.
- b. Diets designed to avoid excessive protein, sodium, calcium, and vitamin D may be of benefit. Excessive restriction or supplementation of dietary phosphorus should probably be avoided. Enhancement of urine volume by feeding a canned diet (and/or a protein restricted diet to dogs to reduce renal medullary urea), and encouraging water consumption may also be of benefit. Although understandably difficult in some patients, fluid intake should be encouraged throughout the day to promote a constantly high urine volume. In humans, high fiber diets have been shown to reduce intestinal absorption and urinary excretion of calcium.
- c. With the exception of Brushite, calcium phosphates tend to be less soluble in alkaline urine. Whether or not such patients would benefit by use of appropriate dosages of acidifiers is unknown. Acidification tends to enhance urine calcium excretion, and is a risk factor for calcium oxalate urolith formation. Pending further studies, we are unable to recommend the routine use of urine acidifiers for patients with calcium phosphate urolithiasis.

To our knowledge, medical dissolution of calcium phosphate uroliths has not been attempted in dogs with distal renal tubular acidosis (RTA). Diets designed to dissolve struvite uroliths would generally not be expected to promote dissolution of calcium phosphate uroliths, in part because they may tend to promote acidemia and aciduria, thus potentially enhancing hypercalciuria and hypocitraturia. However, correction of hypercalciuria, hyperphosphaturia, and hypocitraturia by alkalinization therapy with potassium citrate might promote dissolution of these uroliths in patients with complete or incomplete distal RTA. Chronic alkalinization therapy appears to be beneficial in preventing calcium phosphate urolith formation in human beings with distal RTA. Such therapy has been advocated for patients with complete or incomplete forms of distal RTA because it decreases urolith formation and nephrocalcinosis, and increases urine citrate concentration.

Oral administration of sodium chloride, long recommended for all forms of urolithiasis, may promote hypercalciuria and calcium phosphate urolith formation. Therefore, oral salt therapy is not recommended to promote diuresis in dogs with uroliths containing calcium salts.

All prevention recommendations should be adjusted to meet individual patient's needs. We recommend follow-up urinalyses, serum chemistry profiles, and radiographs on a periodic basis.

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Further references:

<sup>1</sup>Lulich JP, Osborne CA, Unger LK, et al: Nonsurgical removal of urocystoliths by voiding urohydropropulsion. In *Journal of the American Veterinary Medical Association*. Vol 203, pp. 660-663, 1993

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