



A REVIEW ON UROLITHIASIS IN DOGS AND CATS

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Summary

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Urolithiasis is a nutritional disease that affects domestic carnivores. Past and recent literature on urolithiasis was reviewed for information on anatomical occurrence, physiology of urine formation, prevalence, mineral composition, clinical signs, laboratory findings, dissolution therapy, surgery and prevention of urolithiasis. The acquired knowledge of complexed and multifaceted urolithiasis is a tremendous achievement towards the treatment and control of the disease. However, eradication of the disease is the most challenging as it requires total overhaul of all the factors that are responsible for the formation of uroliths.

Key words: cat, dog, urolithiasis, uroliths

INTRODUCTION

Urolithiasis can be defined as the formation of sediment anywhere within the urinary tract which consists of one or more poorly soluble urine crystalloids. An urolith may be defined as the aggregation of crystalline and matrix materials that form in one or more locations within the urinary tract when urine becomes oversaturated with crystallogenic substances, and may be composed of one or more mineral types (Ulrich *et al.*, 1996). One study of 20,343 feline uroliths reported that 5.6% were composed of uric acid and urate. This is consistent with prior studies re-

porting rates of 3.1% to 6.3%. In another study of 5,230 feline uroliths and urethral plugs, stones, which contained a component of urate, were identified in 507 cases (10%) (White, 1996).

HISTORICAL BACKGROUND

The term *urolith* is derived from the Greek *ouron* meaning urine, and *lithos* meaning stone (Osborne *et al.*, 1999). The formation of uroliths specifically in cats and dogs is not a new phenomenon. Ashmont (1891) said of bladder uroliths in

dogs, “a cure was out of the question”. Kirk (1925) described “retention of the urine” as a very common condition in cats, and Blount (1931) also noted that seven different types of uroliths occur in cats, and that magnesium ammonium phosphate was present in the majority of such deposits in alkaline urine. Kidney stone disease (nephrolithiasis) has been a well-known entity for centuries. This has been established by different archeological findings, as well as by writings about painful stone colic and therapeutic trials for stone removal (Eknoyan, 2004).

Although not so well documented, the oldest urolith found in an animal dates back to the Upper Cretaceous age, before the evolution of mankind. The skeleton of a sea reptile, living around 80 million years ago, was found to contain a urolith composed of calcium carbonate and calcium phosphate. Later findings include uroliths found in the bladder of a cave bear and in the kidneys and bladders of sacrificial animals (Hesse *et al.*, 1994). Since then, stones have been found within the urinary tract of many mammals including humans, horses, donkeys, cows, sheep, rabbits, guinea pigs, chinchillas, cats, dogs (Osborne *et al.*, 1989; Weber *et al.*, 2000). Invertebrates such as snails and insects are also known to form concretions in the faecal bladder and the Malpighian tubes, which tend to be fatal (Hesse *et al.*, 1994).

The first documents that testified at the finding of concretions in the kidneys and bladder of animals, which were sacrificed, date back to Herodotus and Aristotle (Anke & Henning, 1973). By 1663 Rofink had classified uroliths according to their size, shape, surface and colour (Schneider, 1985) and by the 18th century, they were recognised to contain uric acid, oxalic

acid, cysteine, and organic materials (Anke & Henning, 1973).

ANATOMICAL OCCURRENCE

Upper urinary tract stones are uncommonly reported in cats and dogs and the vast majority of uroliths (>95%) submitted for analysis are removed from the lower urinary tract (Osborne & Fletcher, 1995). It seems more likely that there is a real species difference. It has been hypothesised that this may, at least in part, relate to the positioning of the kidney and bladder relative to gravity in quadrupeds and bipeds (Markwell *et al.*, 2000). Anatomical differences in the structure of the kidney and in particular the absence of renal pyramids and calyces in the dog and cat kidney, may also contribute to observed anatomical differences in the occurrence of urolithiasis. Urolithiasis accounts for 15% to 23% of cases of feline lower urinary tract disease; up to 11% of cases are due to anatomic defects; and 1% to 8% are urinary tract infections (Gerber *et al.*, 2005). In cats older than 10 years, 46% of lower urinary tract disease causes are related to infection and 17% to concurrent infection and calculi (Bartges & Blanco, 2001).

UROLITH FORMATION

The formation of uroliths involves multiple physiological and pathological processes (Osborne *et al.*, 1996a). An understanding of the processes involved in the formation and elimination of urine is an essential prerequisite for investigations into the role of diet in urolithiasis. The kidneys have two main functions within the body. The primary function is to eliminate waste materials either ingested or produced by metabolism. The second

function is to control the volume and composition of the body fluids. For water and virtually all electrolytes in the body, the balance between intake (due to ingestion or metabolic production) and output (due to excretions or metabolic consumption) is largely maintained by the kidneys. This regulatory function maintains a stable environment for cells allowing them to perform various essential activities (Guyton & Hall, 1996).

Most substances that must be cleared from the blood, especially the end products of metabolism, such as urea, creatinine, uric acid and urates, are poorly reabsorbed and are therefore excreted in large amounts (Guyton & Hall, 1996). Creatinine may be used as a measure of glomerular filtration rate because creatinine appears at the same concentration in the filtrate and the plasma, and is not reabsorbed by the tubules (Finco, 1995). Electrolytes such as sodium ions, chloride ions and bicarbonate ions are highly reabsorbed so that only small proportions of the total amounts filtered appear in the urine. Certain nutritional compounds such as amino acids and glucose are completely reabsorbed in healthy animals and are therefore not usually found in the urine (Guyton & Hall, 1996).

PREVALENCE OF UROLITHIASIS

Three percent (3%) of dogs seen at veterinary hospitals are affected by urolithiasis (Osborne *et al.*, 1995). More specifically, the prevalence of urolithiasis in dogs was reported to be between approximately 0.25 and 0.5% in Sweden and Norway (Wallerstrom & Wagberg, 1992) and 3% in Ukraine (Tion, 2012). Morbidity rates were reported as approximately 0.5% in North American veterinary colleges and between 0.5 and 1% in Germany (Hesse,

1990). Urological diseases constitute a more common reason for the presentation of cats at veterinary hospitals, involving over 7% of the feline case load (Osborne *et al.*, 1995). One study examining obstructed cats found 59% presented with urethral plugs and 12% with uroliths. In the remaining 29%, no specific cause could be ascertained (Osborne *et al.*, 1989). A second study examining non-obstructed cats found urolithiasis in approximately 13% of cases while the underlying cause of urinary tract signs could not be identified in 64% of cases (Buffington *et al.*, 1997). Other causes of the disease included anatomical abnormalities (9%) and behavioural problems (9%). Typically, when causes cannot be identified, cats are said to have idiopathic lower urinary tract disease.

MINERAL COMPOSITION OF UROLITHS

Uroliths can range in size from sand-like material to large individual stones that may grow to fill the entire cavity in which they form. Around twenty crystalline substances can occur in uroliths across all species, made up of various mineral combinations of a few chemical substances, including phosphate, calcium, oxalate, urate, cystine, carbonate and silica (White, 1996). Uroliths that form in cats, dogs and humans can be grouped into four main mineral types, namely urate (including ammonium urate, sodium urate and uric acid), cystine, magnesium ammonium phosphate and calcium (calcium oxalate and calcium phosphate). Uroliths composed predominantly of urate or cystine occur infrequently in dogs and cats, each making up less than 10% of uroliths analysed at one centre in the USA, while struvite (magnesium ammonium phos-

phate) and calcium-containing uroliths are the two most prevalent mineral types found in cats and dogs (Osborne & Fletcher, 1995).

Cystine uroliths

Cystinuria is a congenital disorder characterised by the excretion of excessive amounts of cystine, lysine, ornithine and arginine (Treacher, 1962). Cystine uroliths occur in cats with cystinuria, an inborn error of metabolism characterised by defective proximal tubular reabsorption of cystine and other amino acids (ornithine, lysine, arginine) (DiBartola *et al.*, 1991). No obvious gender or breed predisposition has been reported but Siamese may be at risk (Lekcharoensuk *et al.*, 2001a). Most affected cats are mid age and older (DiBartola *et al.*, 1991). In normal dogs cystine is freely filtered at the glomerulus, and most is actively reabsorbed in the proximal tubule. Cystinuric dogs reabsorb a much smaller proportion of the amino acid from the glomerular filtrate and some may even have net cystine excretion. The main manifestation of this abnormality is the formation of cystine uroliths. Dietary intervention plays a relatively minor role in the management of this type of stone disease, and although cystine solubility can be enhanced by inducing an alkaline urine pH, the solubility does not increase significantly until the urine pH is above 7.5 (Dent & Senior, 1955).

Chemical modification of the cystine molecule into a more soluble form which can be excreted safely in the urine, using drugs such as D-penicillamine (DP) or 2-mercaptopyrionylglycine (2-MPG), is usually required to effectively prevent recurrence in most individuals (Hoppe *et al.*, 1993). Although DP is effective in preventing formation, and may also assist with dissolution of cystine uroliths, this

treatment is accompanied by frequent complications that may limit its use. In humans around 50% of patients will suffer side-effects including skin rashes, neutropenia, thrombopenia, nephrotic syndrome and fever, while severe proteinuria is experienced in 10–15% of patients (Dahlberg *et al.*, 1977). In dogs, the most common side effect is vomiting (Osborne *et al.*, 1995). 2-MPG is related to DP, but has a higher oxidation-reduction potential and may, therefore, be more effective in the disulphide exchange reaction (Keene, 1992). In addition, 2-MPG also tends to result in fewer adverse side effects.

Xanthine uroliths

Xanthine uroliths are rare in dogs and may be due to an inborn error of purine metabolism or administration of allopurinol. In most cases, no identifying risk factors are observed. There is no apparent breed, age or sex predisposition reported. Risk of recurrence is high (within 3–12 months) (White *et al.*, 1996).

Uric acid/urate uroliths

Ammonium urate is the third most common urolith reported in cats. They are composed of uric acid and the monobasic ammonium salt of uric acid (ammonium acid urate) (Westropp *et al.*, 2006). In normal healthy humans, dogs and cats, uric acid is one of several products of purine nucleotide metabolism (Bartges *et al.*, 1999). In turn, uric acid is metabolised by hepatic uricase to allantoin, which is excreted by the kidneys and is readily soluble in the urinary environment (Hoppe *et al.*, 1993). Urate uroliths may occur in cats with portosystemic shunts or any form of severe hepatic dysfunction. This may be associated with reduced hepatic conversion of ammonia to urea resulting in hyperammonaemia. Urate uroliths may

also occur in cats with urinary tract infections leading to increased urine ammonia, in cats with metabolic acidosis and highly acidic urine, and when cats are fed diets high in purines, such as liver or other organ meats (Ling & Sorenson, 1995). The exact pathogenesis in most cats remains unknown in the majority of cases (Westropp *et al.*, 2006). Uroliths containing urate can occur in a number of different forms. In dogs and cats, ammonium urate is the most common form, followed by sodium urate and rarely uric acid. In humans, the form in which urate occurs is largely dependent upon the urine pH during formation. Uric acid crystals tend to form in urine with a pH of <5.5, and within the normal pH range for humans this mineral tends to exist as a mixture of the undissociated ion and free urate (Robertson, 1993).

In dogs and cats, the factors determining the form in which urate occurs is not clearly defined, and often stones containing uric acid and ammonium/sodium urate form. The largest group affected by uric acid/urate urolithiasis is the Dalmatian dog, which only converts 30–40% of uric acid to allantoin (Porter, 1963). This genetically-inherited defect in Dalmatians is thought to be due to impaired transport of urate across the hepatocyte cell membrane, as uricase concentrations tend to be normal (Giesecke & Tiemeyer, 1984). In addition, intestinal uptake of hypoxanthine and urate is delayed (Briggs & Harley, 1986), and renal reabsorption of urate in the proximal tubule is reduced (Roch-Ramel *et al.*, 1976). Dogs and cats with portal-systemic shunts are the second largest group affected. Ammonia is generated by bacterial action on amino acids and urea in the colon (Rothuizen *et al.*, 1982). After intestinal absorption, normal hepatic function converts most portal

blood ammonia to urea so that systemic blood ammonia concentrations remain low. Portal-systemic vascular shunts resulting from congenital malformation or hepatic cirrhosis lead to reduced extraction of ammonia from the portal blood and systemic blood ammonia concentrations become elevated. Normal hepatic conversion of urate to allantoin is reduced and plasma urate concentrations are increased in affected dogs (Hardy & Klausner, 1983). Together these factors result in increased renal excretion of ammonia and urate, with increased risk of subsequent urate urolith formation. In some cases, urate or uric acid uroliths form sporadically in both cats and dogs for reasons remaining unknown. The primary goal for the management of urate urolithiasis is to reduce urinary ammonium and urate activity. In humans, low purine diets have been found to reduce urate production. Severe purine restriction has also been found to reduce urinary urate excretion in both healthy dogs and Dalmatians. However, allopurinol, a drug which decreases the conversion of xanthine to uric acid, by inhibiting the enzyme xanthine oxidase, may also be necessary to prevent recurrence (Senior, 1992).

Magnesium ammonium phosphate and calcium uroliths

The most common component in canine uroliths is struvite (magnesium ammonium phosphate). The formation of struvite uroliths in dogs is closely linked to concurrent urinary tract infection, whereas most feline struvite uroliths are sterile. Manipulation of urine pH 5.5–6.0 is suitable for prevention and dissolution of uroliths (Stevenson *et al.*, 1998). Based on the observation, hypercalciuria, rather than hyperoxaluria, may be a predisposing factor of calcium oxalate urolith formation

in individual dogs and cats. However, in 71% of animals, both urine Ca:Cr and Ox:Cr were comparable with those measured in healthy subjects, which suggests that other urolith promoting factors may be involved as well (Dijcker *et al.*, 2012).

Calcium salts (phosphate and oxalate) and magnesium ammonium phosphate (MAP) are the commonest minerals found in uroliths of cats, dogs and humans (Osborne *et al.*, 1995). In humans, calcium-containing stones predominate with almost one half made up of pure calcium oxalate and the remainder consisting of a mixture of calcium oxalate and calcium phosphate or uric acid or occasionally ammonium urate (Robertson, 1993). Historically, MAP has been the most common type of urolith found in both cats and dogs. However, calcium oxalate-containing uroliths appear to have become more common in both species recently, at least in North America. Recent data from the USA suggest that calcium oxalate-containing uroliths may now be more important numerically than MAP in cats, although the latter still predominate in dogs (Buffington *et al.*, 1997). Calcium oxalate uroliths may be pure, but more commonly present in combination with variable amounts of calcium phosphate, or less commonly MAP or ammonium urate (Osborne *et al.*, 1996b).

In contrast to the situation in dogs, the majority of struvite uroliths in cats are sterile (Lekcharoensuk *et al.*, 2001a). Infection with urease splitting organisms is rare in cats and is identified more often in cats less than a year of age, in older cats, and in cats with compromised host factors (e.g. perineal urethrostomy). Struvite uroliths form when the urine becomes supersaturated with magnesium, ammonium, and phosphorus and when the urine pH is >6.5. Super saturation of the urine with

these minerals can occur with intravascular volume depletion and water conservation. In a case control study, it was shown that diets with the highest magnesium, phosphorus, calcium, chloride and fibre, moderate protein and low fat content were associated with increased risk (Lekcharoensuk *et al.*, 2001b). Diets containing 0.15 to 1.0% magnesium on a dry matter basis have been associated with the formation of struvite uroliths. However, the magnesium effect depends on the form of magnesium and on urine pH. Also, cats fed 0.5% magnesium chloride did not form struvite uroliths whereas cats that were fed 0.5% magnesium oxide did form struvite uroliths. The difference in susceptibility to struvite formation was due to magnesium oxide promoting the formation of alkaline urine whereas magnesium chloride promoted the formation of protective acidic urine (Buffington *et al.*, 1994).

Calcium oxalate

Persistent aciduria may be associated with low-grade metabolic acidosis, which promotes bone mobilisation of carbonate and phosphorus to buffer hydrogen ions. Simultaneous mobilisation of calcium coupled with inhibition of renal tubular reabsorption of calcium, results in increased urinary excretion of calcium (hypercalciuria). Cats fed diets formulated to produce a urine pH between 5.99 and 6.15 were 3 times more likely to develop calcium oxalate uroliths (Lekcharoensuk *et al.*, 2001a). In 5 cats with hypercalcemia and calcium oxalate uroliths, discontinuation of acidifying diets or urinary acidifiers was associated with normalisation of serum calcium concentration (McClain *et al.*, 1999). However, many cats are fed acidifying diets and yet few appear to develop hypercalcemia, metabolic acidosis, and calcium oxalate urolithiasis. There-

fore, additional factors such as gastrointestinal hyper-absorption or increased renal excretion of calcium and/or oxalate may be important in susceptible cats. Increased intestinal absorption of calcium may occur due to excess dietary calcium, excess vitamin D, or hypophosphatemia. Increased renal excretion of calcium may occur with decreased renal tubular reabsorption (furosemide and corticosteroids), or increased mobilisation of calcium from body stores (acidosis, hyperparathyroidism, hyperthyroidism, excessive vitamin D) (Ling *et al.*, 1990).

A case-control study reported that cats fed diets low in moisture and low in protein had an increased risk of calcium oxalate urolithiasis. Cats that consume high protein diets are reported to have increased water consumption, urine volume, and urinary phosphorus excretion, while calcium excretion is not increased. Intravascular volume depletion and concentration of urine volume increases the risk of urine super saturation with calcium and oxalate. Cats fed diets high in moisture content are about one third as likely to develop calcium oxalate uroliths compared to cats fed diets low in moisture (Lekcharoensuk *et al.*, 2001a). High moisture diets are associated with the production of increased volumes of less concentrated urine, compared with the consumption of low moisture diets. Pyridoxine (vitamin B6) increases the transamination of glyoxylate, an important precursor of oxalic acid, to glycine. Therefore, pyridoxine deficiency increases the endogenous production and subsequent excretion of oxalate. A naturally occurring form of this syndrome has not yet been reported. Furthermore, supplementation with vitamin B6 does not decrease urinary oxalic acid excretion compared with a diet containing adequate levels of vitamin B6

(Wrigglesworth *et al.*, 1999). Both dietary magnesium restriction and magnesium supplementation have been associated with increased risk of calcium oxalate urolithiasis in cats; consequently, to minimise calcium oxalate urolithiasis, diets should neither be severely restricted nor supplemented with magnesium (Lekcharoensuk *et al.*, 2001a). Supplemental sodium chloride has long been suggested to increase urinary calcium excretion in humans. However, a recent epidemiological study by Lekcharoensuk *et al.* (2001b) did not support this hypothesis and found that increasing dietary sodium reduces the risk of calcium oxalate uroliths in cats. The risk for calcium oxalate urolith formation increases with age. Smith *et al.* (2004) reported that old cats with mean age 10.63 ± 1.32 years produced urine that had significantly lower struvite relative supersaturation (RSS) values (0.721 ± 0.585 vs. 4.984 ± 4.028) and significantly higher calcium oxalate RSS values (3.449 ± 1.619 vs. 0.911 ± 0.866) when compared to a group of younger (4.06 ± 1.02 years) cats. The old cats had a significantly lower urine pH, compared to the younger cats (6.08 ± 0.22 vs. 6.38 ± 0.22 , respectively). The decrease in urine pH in the old cats may partially explain the increased risk for forming calcium oxalate uroliths with increase in age (Smith *et al.*, 2004). Indoor housing has been reported as a risk factor for calcium oxalate urolithiasis (Gerber *et al.*, 2005).

Calcium phosphate

Calcium phosphate uroliths are uncommon in cats. Hydroxyapatite and carbonate apatite are the most common forms; brushite (calcium hydrogenphosphate dehydrate) is less common. Pure calcium phosphate uroliths may be associated with primary hyperparathyroidism, disorders

that predispose to hypercalciuria (hypercalcemia, excess vitamin D, systemic acidosis, excess dietary calcium), disorders that predispose to hyperphosphaturia (excess dietary phosphorus), decreased urine volume, highly alkaline urine and, at least for nephroliths, the presence of blood clots (Osborne *et al.*, 1995). They often occur as a minor component with struvite and calcium oxalate stones.

CLINICAL SIGNS AND LABORATORY FINDINGS

Haematuria, pollakiuria, stranguria, and dysuria are common clinical signs of lower urinary tract disease and are not specific for cystic calculi. Most cats aged 1 to 10 years with lower urinary tract disease have idiopathic cystitis (55% to 64%).

In the male cat the urethra is blocked and the associated signs are reduction in the quantity or quality of using stream, absence of urine stream and urinary bladder distension signs will develop in cases of urinary obstruction and become increasingly severe with time. If the urinary tract ruptures at any point, evidence of urinary leakage into the surrounding areas may be detected (Drobatz, 2009).

The patient's biochemical profile and complete blood count may be normal. In some cases, abnormalities may suggest a certain urolith type, such as an association of hypercalcemia with calcium oxalate or calcium phosphate uroliths. Azotemia may be present with either upper or lower urinary tract obstruction. Ross *et al.* (2007) reported that it was unclear whether the presence of non-obstructing nephroliths incited renal failure in cats with chronic kidney disease. Uroliths of both the upper and lower urinary tracts may cause secondary infection. Leukocytosis may be seen with pyelonephritis in some cases but is

not associated with simple cystitis (Bartges, 2004).

Urinalysis is an important part of the diagnostic evaluation for all urinary disorders. Crystal solubility is affected by urine pH. Struvite uroliths are more likely to form in alkaline urine; calcium phosphate in alkaline to neutral urine; calcium oxalate and silica in neutral to acidic urine; and urate, xanthine, cystine, and brushite in acidic urine (Adams & Syme, 2005). In patients without urinary tract disease, calcium oxalate and struvite crystals may form in urine samples that have been refrigerated or analysed more than 4 to 6 hours after collection, but in patients with uroliths, crystalluria in a fresh urine sample (<60 min) may provide clues to urolith composition.

Alkaline urine increases the struvite activity product and is commonly found in association with struvite urolithiasis. Crystalluria without stone formation is not pathologic and can be found in healthy animals. *In vitro* crystal formation can occur as a result of prolonged storage, refrigeration, and alkalinisation (Albasan *et al.*, 2003). Crystalluria can be confirmed by evaluation of fresh urine samples. Pyuria may result from concurrent infection. Urine culture via cystocentesis is recommended to detect bacterial infection (Hostutler *et al.*, 2005).

Urine sediment examination may reveal pyuria or bacteriuria. Urine culture is indicated in all cases of urolithiasis. Infection has been documented in 75% of dogs with cystic calculi when the results of urine, bladder mucosal biopsy, and urolith culture are combined (Gatoria *et al.*, 2006). Specialised tests may be recommended for specific urolith types (e.g., tests for hyperadrenocorticism in patients with calcium oxalate uroliths).

DIAGNOSTIC IMAGING

Not all patients with signs of urinary disease need imaging at first presentation. A female dog with a short duration of polakiuria and urgency may have a simple bladder infection that will resolve with a short course of antibiotics. For dogs and cats with lower urinary tract signs, imaging is recommended if clinical signs do not resolve rapidly or if signs recur rapidly or frequently and the breed is predisposed to urolithiasis. Because nephrolithiasis and ureterolithiasis are increasingly documented in cats with chronic kidney disease, radiography is recommended for all cats with diagnosed chronic kidney disease (Ross *et al.*, 2007). Radiography is a sensitive test for detection of struvite calculi, which are radiopaque. If the calculi are small (<3 mm), ultrasonography or double-contrast cystography is superior to radiography for detection (Hostutler *et al.*, 2005).

Survey radiography

Most opacity seen in the urinary bladder on survey radiographs are calculi. Other possibilities include mineralised neoplasia and mucosal wall mineralisation (Park & Wrigley, 2007). Calcium oxalate and struvite uroliths are generally radiopaque; however, 1.7% to 5.2% of these uroliths are not apparent on survey radiographs. These undetected uroliths are usually small (<1 mm). Urate, cystine, and calcium phosphate calculi are variably radiopaque, and approximately 25% of survey radiographs are interpreted as negative for these uroliths. The incidence of false negative results with survey radiography is 13% for all urolith types (Weichselbaum *et al.*, 1999). Urethral calculi are easily overlooked, particularly if the entire urethra is not included in the view. To avoid confusion with the pelvic limbs in

male dogs, it may be helpful to pull the pelvic limbs forward to get an unobstructed view of the urethra. Mineral opacity in the kidneys may be caused by renal calculi, calcification of the renal parenchyma (nephrocalcinosis), calcified tumours, or mineralised cysts. Most mineral opacities in the area of the ureter represent calculi. The end-on view of the deep circumflex iliac artery can be mistaken for a distal ureteral lesion (Feeney & Johnston, 2007). Nipples and other structures can also be mistaken for uroliths. Lateral and dorsoventral radiographic views should be compared. Small ureteral calculi or those overlying colonic content may be overlooked; the sensitivity of survey radiography for detecting ureteroliths in cats is 81% (Kyles *et al.*, 2005).

Contrast radiography

Pneumocystography (negative-contrast cystography) is more sensitive than survey radiography at detecting calculi, with a false-negative rate of 6.5%. Double-contrast radiography, in which the bladder is distended with gas and contrast medium is instilled (approximately 1 to 3 mL for a cat or small dog and 3 to 5 mL for a large dog), further improves diagnostic accuracy for urolith detection, with a false-negative rate of 4.5% (Weichselbaum *et al.*, 1999). For calculi detection, a pool of 200 mg/mL of contrast agent (one part contrast to one part saline) 5 mm deep (about 1 to 5 mL) provides the best accuracy for determining the presence or absence of calculi (Feeney *et al.*, 1999).

Although double-contrast radiography is the most sensitive method of counting calculi, an accurate count is obtained in only 53% of cases (Weichselbaum *et al.*, 1999). Excretory urography, also referred to as intravenous pyelography, aids in the diagnosis of upper urinary calculi (Feeney

& Johnston, 2007). An abrupt termination of contrast within the ureters with lack of distal enhancement and dilation of the renal pelvis and ureter signifies obstructive disease. However, filling defects (lack of contrast) within the renal parenchyma or collecting system can also be seen with cysts, neoplasia, granulomas, abscesses, and haematomas. Antegrade pyelography involves ultrasonographic or fluoroscopic guidance to directly inject contrast material into a dilated renal pelvis. Antegrade pyelography may provide a superior image of the renal collecting system and ureters compared with excretory urography (Adin *et al.*, 2003). Contrast medium can interfere with urine culture results and artificially increase protein concentration and urine specific gravity. Urinalysis should not be performed within 24 h after contrast urography (Feeney *et al.*, 1980).

Abdominal ultrasonography

Both radiopaque and non-radiopaque cystic calculi are generally seen with ultrasonography. The interface between the urine and the calculus is intensely hyperechoic (bright white) with acoustic shadows (dark areas) below the calculus. Urethral calculi are difficult to visualise with ultrasound unless they are lodged near the neck of the bladder. Although ultrasonography is better than survey radiography for detecting uroliths, its false-negative rate of 3.5% is similar to that of double-contrast radiography. Renal calculi are usually visible as intense hyperechoic foci with strong acoustic shadowing. However, visualisation may be obscured by overlying bowel gas, and renal calculi may be confused with renal parenchymal calcification or normal shadowing of the collecting system. Pelvic or ureteral dilation makes calculi easier to detect (Nyland *et al.*, 2002). The sensitivity of ultrasono-

graphy for detecting ureteral calculi is 77%, which can be increased to 90% by using a combination of ultrasonography and survey radiography (Kyles *et al.*, 2005).

The presence of uroliths in the urinary bladder of a 3-year-old cat in comparison with that of 7-year-old cat may suggest that both young and adult cats, can come down with urolithiasis. Tion (2012) had earlier reported that cats within the age range of 6–15 years are more susceptible to urolith formation than cats within the age range of 0–5 years. They also reported that the prevalence of urolithiasis in cat is 6% and that nutrition is the greatest factor that predisposes cat to urolithiasis, followed by age (Tion, 2012).

Computed tomography

Uroliths can easily be distinguished from surrounding soft tissue using computed tomography. A recent human study showed that non-contrast computed tomography could be used to predict the composition of uroliths on the basis of differences in radiodensity, measured in Hounsfield units. One *in vitro* study of uroliths removed from dogs showed the prediction of mineral content to be 75% to 88% accurate for pure uroliths, but mixed uroliths could not be accurately assessed (Pressler *et al.*, 2004).

UROLITHIASIS TREATMENT

Dissolution therapy

Dissolution therapy is effective in management of sterile feline struvite calculi. The key goals of dissolution diets include reduction in urine pH to ≤ 6.3 and reduction of dietary magnesium (Osborne *et al.*, 1996a). Osborne *et al.* (1996b) found a mean dissolution time of 36 days (range:

14 to 141 days) for sterile uroliths and 44 days (range: 12 to 92 days) for infected uroliths when Hill's Prescription Diet (Hill's Pet Nutrition) was used. Another commercially available dissolution diet (Medi-Cal Dissolution Formula Veterinary Medical Diets, Guelph, Ontario) was effective in 79% of cases with clinical suspicion for sterile struvite (Osborne *et al.*, 1996b). Treatment failure has been noted with mixed-composition calculi, owner noncompliance, and food refusal (Houston *et al.*, 2004). Dissolution diets are not recommended for growing cats or for cats that are acidemic, pregnant, or hypervolemic because these diets are protein restricted and acidifying and can result in volume expansion from increased sodium concentration (Osborne *et al.*, 1996a). If a calculolytic diet is contraindicated in pregnancy and immaturity or obstruction is present, physical removal of stones via surgical or nonsurgical techniques is recommended. Additionally, if infection is present, dissolution therapy is ineffective without antibiotic therapy. It is recommended that antibiotics be continued 1 month after radiographic dissolution, as bacteria can be released from calculi during therapy or can persistently colonise the uroendothelium and result in relapse (Osborne, 1996a,b). Infection-induced struvite uroliths generally take longer to dissolve than sterile struvite uroliths (Osborne, 1996c). In cats, nephroliths and uroliths are rarely composed of struvite and mostly composed of calcium oxalate. However, unlike feline struvite cystoliths, which are predominantly sterile, most feline struvite nephroliths (80%), are associated with urine culture results which are positive for urease-producing organisms (Kyles *et al.*, 2005). After definitive therapy, routine monitoring using urinalysis and abdominal radi-

ography is recommended. Early detection of recurrence may allow nonsurgical therapies to be used. Cats with risk factors for urinary tract infection (i.e. chronic kidney disease, polyuria, diabetes mellitus, hyperthyroidism, perineal urethrostomy) should have urine samples cultured every 3 to 6 months (Mayer-Roenne *et al.*, 2007). Monitoring urine pH is recommended to assess dietary compliance and efficacy. Values between 6.0 and 6.5 may reduce the incidence of calcium oxalate and struvite crystal formation. Monitoring urine specific gravity has been recommended to assess water consumption. A urine specific gravity <1.030 has been recommended as a goal (Westropp & Buffington, 2005).

Surgery

With the advances in extracorporeal shock wave lithotripsy (ESWL) and endourological surgery, such as uteroscopy (URS) and percutaneous nephrolithotomy (PNL), during the past 20 years, the indications for open stone surgery have markedly diminished (Preminger *et al.*, 2007). Centres with the equipment, expertise and experience in the surgical treatment of renal tract stones reported a need for open surgery in 1–5.4% of cases (Wolf, 2007). Nowadays, the indications for open surgery for stone removal include: complex stone burden; treatment failure with ESWL and/or PNL, or failed ureteroscopic procedure; intrarenal anatomical abnormalities: infundibular stenosis, stone in the caliceal diverticulum (particularly in an anterior calyx), obstruction of the ureteropelvic junction, stricture; morbid obesity; skeletal deformity, contractures and fixed deformities of hips and legs; comorbid medical disease; concomitant open surgery; non-functioning lower pole (partial nephrectomy), non-functioning

kidney (nephrectomy); patient choice following failed minimally invasive procedures, i.e. single procedure in preference to possibly more than one PNL procedure; stone in an ectopic kidney where percutaneous access and ESWL may be difficult or impossible; cystolithotomy for giant bladder calculus and a large stone burden in children because of easy surgical access and the need for only one anaesthetic procedure. Open surgery for renal tract stones has become almost obsolete, with laparoscopic surgery increasingly used in situations for which open surgery would previously have been used, including complex stone burden, failed previous ESWL and/or endourological procedures, anatomical abnormalities and morbid obesity. (Abreu & Gill, 2005). Laparoscopic surgery was initially used for ablative surgery in renal cancer and correction of pelvi-ureteric junction obstruction, but is now being used to remove both renal and ureteric stones. Although, there are anecdotal reports of successful anatomic nephrolithotomy. It is in the removal of ureteric stones that laparoscopy appears to have found its place (Preminger *et al.*, 2007). Ultrasonography and radiography are complementary to each other where prediction can serve as an alternative to distinguish urolith mineral composition whenever performing surgery is difficult to retrieve the urolith in an unstable patient to choosing medical treatment (Fromsa *et al.*, 2011). Laparoscopic cystotomy was used for removal of uroliths from three dogs, but one dog had recurrence of urolithiasis, attributed to inadequate conservative treatment and to the lack of inappropriate diet. It was submitted to another similar videolaparoscopic cystotomy without complication. The proposed technique is appropriate and an alternative to conventional cystotomy for treatment of

canine vesical urolithiasis (Brun *et al.*, 2008). However, spayed females had an increased risk for urolithiasis than intact females (McKenzie, 2010). Because of the risk of surgical damage to functional kidney tissue during surgery, if the nephroliths are presumed to be calcium oxalate and not associated with obstruction of urine flow, uncontrollable infection, or deterioration of renal function, monitoring kidney stone activity and selecting protocols to minimise urolith growth may be the best course of action. Monitor the status of the patients nephrolith activity by radiography or ultrasonography every 2–6 month, unless clinical signs mandate more frequent evaluation (Koehler *et al.*, 2008). Urethral hydropulsion followed by cystotomy can be adopted to remove large cystic calculi and small urethral calculi (Blount, 1931). Urethrotomy is made into the urethral lumen to remove or relieve an obstruction temporarily. When permanent diversion of urine flow proximal to an obstructed, severely damaged, or diseased urethra is required, an urethrostomy is performed. Urethrostomy is also performed to diminish the risk of urethral obstruction due to recurrent urinary calculi. Patients that have an elective urethrostomy have a favourable prognosis if attention is paid to proper surgical technique (i.e. urethral mucosa is sutured to the skin). Scrotal urethrostomy in male dog and perineal urethrostomy for male cats when performed anatomically and with care, provides satisfactory therapies that repeatedly have urethral obstruction (Igna, 2008).

PREVENTION OF UROLITHIASIS

A general recommendation for prevention of urolithiasis is to increase water consumption to encourage diuresis and reduce

time for aggregation and crystallisation. This may be most effectively completed with a transition to a moist diet; however, providing flavoured or running water may encourage increased water intake (Westropp & Buffington, 2005). Dietary therapy may reduce calculi recurrence, although clinical studies on recurrence rates are lacking. Epidemiologic studies suggest that a urine pH of approximately 6.0 to 6.3 and consumption of a low-magnesium diet reduce recurrence of naturally occurring sterile struvite urocystoliths (Osborne *et al.*, 1999). However, acidification of the urine to <6.29 may increase the risk of calcium oxalate urolith formation. Dietary analysis and quantification of “alkalogenic” and “acidifying” components may aid in predicting urine pH but cannot be recommended at this time (Markwell *et al.*, 1998). Urinary acidifiers (DL-methionine, ammonium chloride) should be considered only when the urine pH is >6.5 with *ad libitum* feeding conditions (Westropp & Buffington, 2005). Urinary acidifiers alter the urine pH and SAP but cannot reduce the organic fraction responsible for the matrix formation (Funaba *et al.*, 2001). Control of urinary tract infection and change in diet can help in prevention of urolithiasis in dogs and cats (Bartges, 2013). Nutritional management is the best preventive strategy against urolithiasis. As such it may not replace the surgical procedures but may surely help in decreasing the recurrence rate of uroliths, since long-term pharmacological therapy and its potential side effects often lead to subsequent failure (Samal *et al.*, 2011). All prevention recommendations should be periodically monitored to meet individual patient’s needs. This typically includes follow-up urinalysis, serum chemistry profiles and radiography. Early detection of small urocystoliths that recur

despite appropriate medical therapy facilitates non-surgical removal by voiding urohydro-propulsion (Kochler *et al.*, 2008).

CONCLUSION

Nutrition is the major factor responsible for formation of uroliths in dogs and cats. Since clinical and laboratory parameters of the affected animals can lead to definitive diagnosis of urolithiasis, prevention and treatment of the disease are possible. However, eradication of the disease is the most challenging as it requires total examination of all the factors that are responsible for the formation of uroliths.

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