Under Pressure:
A Case of Canine Urolithiasis and Its Complications

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Class of 2018
Clinicopathological Conference
Presented on October 20, 2017

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Introduction

Urolithiasis refers to the formation of calculi in the urinary tract.[54] These calculi, also called stones or uroliths, are not the result of a single disease process, but rather the consequence of familial, congenital, or acquired pathophysiological factors that, in combination, lead to the oversaturation of urine with lithogenic substances.[11,50,56,68] The resulting uroliths are composed of crystalline aggregates and an organic matrix of non-crystalline proteins.[34] This report details a case of infection-induced magnesium ammonium phosphate (struvite) urolithiasis in a canine patient who was presented to the Mississippi State University College of Veterinary Medicine (MSU-CVM) - Animal Health Center for urethral obstruction. Following is a discussion of the history and presentation, pathophysiology, diagnostic approach and considerations, and treatment and management of urolithiasis. Special attention is given to infection-induced struvite urolithiasis and bacterial cystitis, as well as potential complications, such as urethral obstruction, urinary tract rupture, uroabdomen, and peritonitis.

History and Presentation

Ally, a 9-year-old female spayed Miniature Schnauzer, was presented to the MSU-CVM small animal surgery service on November 2, 2016, for a twenty-four-hour history of anorexia, lethargy, and vomiting. Her owners were unsure of when Ally last urinated. Abdominal radiographs taken by the referring veterinarian revealed a distended urinary bladder filled with numerous uroliths, as well as a stone in the proximal urethra. Cystocentesis was performed prior to referral, removing approximately 300 mL of urine. The remainder of Ally's history was unremarkable, with no previous medical issues.

On presentation, Ally was quiet, alert, and responsive. She weighed 9.7 kg with a body condition score of 7 out of 9. Her temperature (102.2° F), pulse (102 beats per minute), and

respiratory rate (panting) were all within normal limits. Ally's mucous membranes were pink and tacky, with a capillary refill time of 3 seconds. These findings, along with a delayed skin tent, suggested that Ally was approximately 5% dehydrated. Cardiothoracic auscultation was unremarkable. Ally was painful upon abdominal palpation and had a notably distended urinary bladder. Rectal examination revealed a stone in the urethra at the level of the pelvic brim. The remainder of the physical examination was unremarkable.

Ally's history and presentation were consistent with urolithiasis. Patients with urolithiasis are often presented with clinical signs of lower urinary tract disease. The lower urinary tract consists of the urinary bladder and the urethra, as well as the prostate in male dogs, and is the location of the vast majority of urinary calculi.[1,29,30,34,50,56] Dysuria, hematuria, pollakiuria, and stranguria are hallmark signs of lower urinary tract disease and are most often due to cystitis, urolithiasis, or neoplasia of the lower urinary tract.[2,20,30,32,34,40,49,,56,58] In the case of urolithiasis, these signs are likely due to damage of the urinary epithelium, resulting in inflammation.[49] Often no signs are present when calculi are located in the kidneys, unless there is urinary tract obstruction or concurrent infection.[35,46] Signs associated with urinary tract infection include those mentioned above, as well as polydipsia, polyuria, urinary incontinence, discharge from the external genitalia, and malodorous urine.[8,35,58]

Patients who are presented for urethral obstruction frequently have a very distended urinary bladder and are painful upon abdominal or lumbar palpation, as demonstrated in the case of Ally.[30,32,34,57] A calculus may be palpable in the urethra during rectal examination.[32,52] Post-renal azotemia is a common complication of urethral obstruction and often results in anorexia, depression, lethargy, and vomiting.[30,32,34,57] If urethral obstruction has been present for greater than twenty-four hours, other signs of systemic illness may be observed on physical examination.

These signs include dehydration, bradycardia, tachypnea, and hypothermia. Patients may also be weak, stuporous, or laterally recumbent.[57]

In the event of urinary tract rupture secondary to chronic severe urolithiasis or urethral obstruction, patients may present with peritonitis due to either non-septic or septic uroabdomen. Signs of peritonitis are often similar to systemic signs seen with urethral obstruction, such as abdominal distension and pain, depression, vomiting, and diarrhea. As septic peritonitis progresses, other signs may be associated with shock or septicemia. In early septic shock, patients often have red injected mucous membranes, a rapid capillary refill time (less than 1 second), tachycardia, tachypnea, and bounding pulses. As the patient begins to decompensate, clinical signs may include pale mucous membranes, a prolonged capillary refill time, refractory hypotension, and poor peripheral pulses. These signs may progress to multiple organ failure and death.[38,41,43,51]

Pathophysiology

In general, uroliths have four layers: nidus, stone (urolith body), shell, and surface crystals.[3,20] The innermost layer, the nidus, begins to form through a process called nucleation when the urine is supersaturated with lithogenic substances such as salts and crystals. Urine pH, organic matrix proteins, and the balance of crystallization promoters and inhibitors contribute to the sustained growth of the other layers of the urolith around the nidus. [29,34] Diet, endocrinopathies, genetics, medications, metabolic derangements, urinary tract infection, and vascular anomalies are all predisposing factors to urolith formation. [14,29,34] Presence of foreign bodies in the bladder, such as suture material, may also contribute to the formation of calculi.[14]

Uroliths may be composed of one or more types of inorganic or organic crystalloids. The most common mineral composition of stones has long been considered to be magnesium

ammonium phosphate (struvite), followed closely by calcium oxalate; however, many sources report a change in the distribution of uroliths submitted for stone analysis over the past two to three decades, with calcium oxalate becoming the new most common mineral type.[1,5,19,21,25,28,36,50,53,63,65] Other less common mineral types include urate, cystine, calcium phosphate, silica, and xanthine.[25,29,34,50,53,56,63] Upwards of 73% of stones may contain more than one mineral type.[25] Uroliths composed of 70% or more of a single mineral type are often identified by that mineral. Stones lacking a nidus or shell or that contain less than 70% of a single mineral type are classified as mixed, with those containing a core or outer layer of 70% or more of one mineral type with an opposing outer layer or central core of a different mineral type classified as compound.[3,21,28,46,63] Factors affecting the mineral composition of stones include age, breed, sex, anatomic location of the stone, urine pH, and urinary tract infection. [29,34]

For the purpose of this discussion, Ally's stone type is being considered one of magnesium ammonium phosphate; however, stone analysis revealed that her uroliths were composed of two different mineral types: struvite and calcium phosphate carbonate (see Diagnostic Approach and Considerations). Struvite uroliths can be divided into those of sterile origin and those resulting from infection. Stones formed in sterile urine are rarely reported in dogs.[3,29,34,61] The exact cause of sterile struvite urolithiasis is unknown.[34] Most magnesium ammonium phosphate stones in dogs are associated with urinary tract infection.[3,17,29,34,52,61] Urinary tract infection occurs when a breakdown in host defense mechanisms allows an infectious agent to adhere, multiply, and persist within the urinary tract. This agent can be bacterial or fungal.[8] In dogs, the primary manifestation of urinary tract infection is bacterial cystitis, which usually results from bacteria entering the bladder via the urethra.[30] *Escherichia coli* is the most common agent isolated from the bladder. Other organisms include

Corynebacterium spp., *Enterobacter* spp., *Enterococcus* spp., *Klebsiella* spp., *Pasteurella* spp., *Proteus* spp., *Pseudomonas* spp., *Staphylococcus* spp., and *Streptococcus* spp.[8,18,34,42,48]

Struvite urolithiasis is often caused by an infection with one or multiple urease-producing bacteria. Various *Staphylococcus* spp., including *S. intermedius*, and *Proteus* spp. are the most commonly associated pathogens, with some *Corynebacterium* spp., *Enterococcus* spp., *Klebsiella* spp., *Pseudomonas* spp., and *Ureaplasma* spp. also being credited for the production of infection-induced struvite uroliths.[3,19,29,34,42,52,61] Urease, an enzyme that breaks down urea, causes the release of ammonia and carbon dioxide. As ammonia undergoes hydrolysis, ammonium and hydroxyl ions are produced, decreasing hydrogen ion concentrations in the urine. The resulting alkaline urine decreases struvite solubility.[19,34,52] Production of bicarbonate ions also contributes to the increase in urine pH.[29,42,61] Supersaturation of the urine with ammonium, magnesium, and phosphate ions allows for the formation of struvite crystals.[3,29,34,42,52,61] Ammonium also damages the coating of the urinary epithelium, allowing for struvite crystals and bacteria to adhere and aggregate to form uroliths.[61]

Due to their short, wide urethras (relative to male dogs), female dogs are at an increased risk of developing a urinary tract infection.[21,30,37] Therefore, females are much more commonly affected with struvite urolithiasis than males.[21,27,28,29,34,37,53,61,65] While any age can be affected, the most commonly reported range is 3 to 9 years old.[21,28,37,40,65] The most commonly affected breeds include Bichon Frisé, Chihuahua, Cocker Spaniel, Dachshund, Dalmatian, German Shepherd Dog, Golden Retriever, Jack Russell Terrier, Lhasa Apso, Miniature Schnauzer, Miniature Poodle, mixed breed, Pekingese, Pomeranian, Pug, Shih Tzu, Springer Spaniel, and Yorkshire Terrier.[19,21,25,28,34,37,40,53,61,63]

As mentioned above, Ally's stones consisted of both magnesium ammonium phosphate and calcium phosphate carbonate. Pure calcium phosphate stones are rarely reported in dogs.[53,60,63] Most often, calcium phosphate is found as a component of struvite uroliths, as in Ally's case. This observation is likely due to a similar pathogenesis of the two stone types.[21,34,53,56,60,63]

The presence of calculi in the lower urinary tract, regardless of stone type, can lead to various complications. One such complication that was observed in Ally is urethral obstruction. Urinary outflow obstruction leads to the accumulation of urine in the bladder and urethra, resulting in increased intravesicular pressure. Prolonged over-distension of the lower urinary tract leads to bladder atony, mucosal injury, and pressure necrosis of the bladder. The kidneys and ureters may also be damaged by the increased pressure.[10,57] If the urinary bladder ruptures secondary to urethral obstruction, leakage of urine into the abdominal cavity can occur.[59] Uroabdomen is one cause of peritonitis, which can be either sterile or septic. In the case of bacterial cystitis, rupture of the urinary tract can lead to a secondary septic peritonitis.[41,51] Bacteria most commonly associated with septic peritonitis include *Escherichia coli*, *Clostridium* spp., and *Enterococcus* spp.[51] In the case of Ally, the bacteria were likely representative of those causing her cystitis. As discussed earlier, septic peritonitis can lead to systemic signs of illness associated with shock. Shock is defined as inadequate cellular energy production or the inability of the body to supply cells with oxygen and nutrients and remove waste products.[38,39] In the face of a systemic infectious insult or sepsis, septic shock may occur. Septic shock refers to acute circulatory failure and persistent arterial hypotension in the face of sepsis.[38] Initially patients present in the hyperdynamic phase with peripheral vasodilation; however, as physiologic compensation begins to fail, patients decompensate and enter the hypodynamic phase. Without

rapid intervention, shock will progress to cell death, multiple organ failure, and death of the patient.[39]

Diagnostic Approach and Considerations

Initially following physical examination of Ally, a venous sample of blood was drawn for blood gas analysis, CBC, and chemistry. Significant findings revealed by blood gas analysis included a decreased PCO_2 (29.3 mmHg), a decreased BEecf (-8), and a decreased HCO_3 (17.2 mEq/L), with a normal pH (7.377). These findings were suggestive of a compensated metabolic acidosis; however, possible lactic acidosis secondary to shock could produce similar results if offset by hyperchloremic alkalosis from sustained vomiting.[33] CBC revealed a leukopenia (4.0 K/uL, with a reference interval of 7.0-22.0), thrombocytopenia (7 K/uL, with a reference interval of 160-650 and an estimated platelet count of 112), neutropenia (2600 /uL, with a reference interval of 3500-14,200), monocytopenia (80 /uL, with a reference interval of 175-1700), and eosinopenia (40 /uL, with a reference interval of 120-1300). Other findings on CBC included rubricytosis (nucleated RBC 107/100 WBC), moderate megaplatelets, slight polychromasia, slight anisocytosis, slight poikilocytosis, and occasional Howell-Jolly bodies. Azotemia was indicated on chemistry by an elevated BUN (86 mg/dL, with a reference interval of 8-24) and creatinine (2.34 mg/dL, with a reference interval of 0.50-1.40). There was also a decreased CO_2 (17.4 mEq/L, with a reference interval of 20.0-28.0) and an elevated phosphorus (5.8 mg/dL, with a reference interval of 2.5-5.0).

Ultrasonography revealed a distended urinary bladder and urethra, as well as the presence of uroliths in both locations. Abdominal radiographs confirmed the presence of numerous stones in the bladder, as well as one in the proximal urethra and distension of the lower urinary tract. Uroliths were also noted in the left and right kidneys, and the liver appeared to be enlarged with

a rounded margin. Following sedation, a red rubber catheter was passed to unblock the urethra and drain the bladder. A Foley catheter was then passed in Ally's urethra prior to taking a second set of abdominal radiographs, confirming resolution of urethral obstruction. The urine collected from catheterization was submitted for urinalysis and urine culture. The urine was red and cloudy with 3+ protein and 4+ SSA, moderate bilirubin, hematuria (RBC too numerous to count), pyuria (5-10 WBC), and rare bacteria. Results of the urine culture were reported on November 5, with growth of *Enterococcus faecalis, Klebsiella pneumoniae* ssp. *pneumoniae,* and *Staphylococcus intermedius*.

On November 3, 2016, a repeat CBC and chemistry were performed, as well as a reticulocyte count to investigate the rubricytosis observed on initial bloodwork. Results of the CBC included a leukopenia (4.8 K/uL), thrombocytopenia (4 K/uL, with an estimated platelet count of 80), and neutropenia (2592 /uL). Other findings on CBC included rubricytosis (nucleated RBC 19/100 WBC), moderate megaplatelets, slight polychromasia, slight anisocytosis, slight poikilocytosis, and rare to slight Howell-Jolly bodies. Chemistry revealed a resolution of azotemia, with an elevated chloride (126.0 mmol/L, with a reference interval of 106.0-122.0) and a decreased albumin (2.0 g/dL, with a reference interval of 2.5-3.9). The reticulocyte count was elevated (2.9%, with a reference interval of 0.0-1.0), suggestive of a regenerative response.

Results of bladder mucosal culture following Ally's surgery on November 3, 2016, (see Treatment and Management for discussion of Ally's surgery) were reported on November 6, and confirmed infection with the same bacteria cultured from the urine. Stone analysis results were reported on November 21, revealing a urolith body of 55% magnesium ammonium phosphate

and 45% calcium phosphate carbonate. The shell was 95% struvite and 5% calcium phosphate. Post-operative radiographs confirmed removal of the uroliths from the urinary bladder.

Similar to the initial diagnostic approach of Ally's case, complete blood count, chemistry, and urinalysis should be performed whenever a patient presents with signs of lower urinary tract disease. While CBC can often be normal in the case of urolithiasis or simple cystitis, signs of a more severe infection, such as leukocytosis, may be noted.[56] Azotemia is a common chemistry finding and may be pre-renal, renal, or post-renal, depending on whether the upper or lower urinary tract is affected. Urinalysis often reveals similar results for urolithiasis and bacterial cystitis. Findings may include bacteriuria, crystalluria, proteinuria, pyuria, and an alkaline pH.[8,61] Struvite crystals have a coffin lid appearance.[61] Their presence can be a normal finding in healthy patients and, even in the presence of urolithiasis, may not be reflective of the stone type. Refrigeration of urine samples can also lead to the formation of struvite crystals.[64]

In addition to bloodwork and urinalysis, urine culture is indicated in the case of urolithiasis, as upwards of 75% of dogs with stones in the bladder may have a concurrent urinary tract infection.[56] There is some evidence to support that if a positive culture is obtained from urine collected by cystocentesis, then no other samples are required for culture and sensitivity.[18,48] However, if uroliths are removed surgically, it is generally recommended to collect a bladder mucosal biopsy for culture, as the bacteria present may differ from those found in urine.[4,18,42]

Survey radiography is one of the most useful methods for diagnosis of urolithiasis.[2] Radiopaque uroliths, such as magnesium ammonium phosphate, calcium phosphate, and calcium oxalate, are usually detectable on radiographs.[16,29,62] Stones of mixed or compound composition are also likely to be visible.[16] Cystine, urate and xanthine calculi are considered radiolucent, and

therefore, are often missed on radiographs.[16,29,56,62] Small uroliths may also go unnoticed by survey radiography.[29,56] In these instances, double-contrast and negative contrast cystography, ultrasonography, and computed tomography are more sensitive imaging modalities for the detection of uroliths.[9,22,29,46,56]

Despite the usefulness of the diagnostic approaches above for detecting the presence of urolithiasis and bacterial cystitis, a method to accurately determine urolith composition *in vivo* has yet to be developed.[46] Some stones have relatively characteristic appearances and can be subjectively evaluated to determine mineral type.[29] Struvite calculi are often white, yellow-white, yellow, red, or gray in color.[53] They tend to be faceted or pyramidal in shape, with smooth, blunt margins.[53,69] Struvite calculi are on average larger than those of other mineral types. In fact, the larger the stone, the more likely it will be composed of magnesium ammonium phosphate.[53] Unfortunately, the only true means of definitive diagnosis of stone type is via chemical or physical stone analysis, with physical analysis being the more reliable method. Voided stones and those that are manually removed by surgery or less invasive measures can be submitted to a diagnostic laboratory for analysis.[29] Uroliths submitted for analysis should not be placed in formalin when shipped, as this may lead to misdiagnosis of mineral composition.[12,61]

While Ally's urethral obstruction was simply diagnosed by the presence of a stone in the proximal urethra and distension of the lower urinary tract on survey radiography and abdominal ultrasonography, other findings may also be suggestive of outflow obstruction. CBC may reveal a leukocytosis, and chemistry findings can include azotemia, hyperglycemia, hyperkalemia, hyperphosphatemia, hypocalcemia, and hyponatremia. Serum bicarbonate and pH may be decreased, indicating metabolic acidosis. Bacteriuria, crystalluria, casts, glucosuria, hematuria, and proteinuria are possible findings of urinalysis. Electrocardiography may reveal signs of

hyperkalemia, such as a prolonged PR interval, decreased to absent P waves, widened QRS complexes, short QT intervals, and tall T waves.[57]

In the event of urinary tract rupture, diagnostic imaging may not provide a straightforward answer, as an intact bladder does not rule out leakage from other parts of the urinary tract.[41] Evaluation of abdominal fluid is much more useful for confirming urinary tract rupture by identifying the presence of uroabdomen. Abdominal fluid creatinine that exceeds 2 times that of serum creatinine, as well as abdominal fluid potassium that exceeds 1.4 times that of serum potassium, is suggestive of uroabdomen.[55] Septic peritonitis can be diagnosed by similar means; an abdominal fluid glucose concentration that is 20 mg/dL lower than that of serum glucose is representative of septic peritonitis in dogs. Abdominal fluid lactate that is at least 2 mmol/L or higher than blood lactate also confirms septic peritonitis.[43,51,55] The presence of bacteria in the abdominal fluid is also indicative of septic peritonitis.[103] Collected fluid should always be cultured for definitive diagnosis and to direct antibiotic treatment choices.[55]

CBC, chemistry, and urinalysis may also be useful in identifying cases of septic peritonitis. Initial CBC findings often include a neutropenia and thrombocytopenia.[43] As the peritonitis progresses, neutrophilia with a left shift is a more common lab result. Anemia and polycythemia are also possible findings on complete blood count. [43,51] Chemistry findings can include an increased ALT and AST, azotemia, hyperbilirubinemia, hyperkalemia, hypoglycemia, and hypoproteinemia. An increased urine specific gravity may be observed by urinalysis. Other diagnostic findings associated with septic peritonitis include hypotension and arrhythmias on electrocardiography.[51]

Treatment and Management

On presentation, Ally was administered a quarter shock dose of LRS (approximately 220 mL) over roughly 15 minutes. As mentioned earlier, a Foley catheter was placed in Ally's urethra. She was monitored overnight in the MSU-CVM ICU and maintained on methadone (0.1 mg/kg IV q6h), Unasyn (30 mg/kg IV q8h), and LRS (51 mL/h IV or approximately double maintenance).

Ally was scheduled for a cystotomy on November 3, 2016. Under general anesthesia, she was placed into dorsal recumbency, and her abdomen was clipped and prepped with isopropyl alcohol and 4% chlorhexidine scrub. Using a #10 blade, an approximately 9 cm long skin incision was made along midline spanning from the xiphoid bone to the pubis bone. A combination of blunt and sharp technique was used to dissect through fat and subcutaneous tissue down to the body wall. Monopolar cautery was used for hemostasis. The linea alba was tented upwards with thumb forceps, and a stab incision was made into the body wall with a #15 blade. The incision was extended cranially and caudally along the length of the initial skin incision, and the falciform fat was removed using cautery. Moistened lap sponges were placed on the cranial, caudal, and lateral margins of the incision, and a Balfour retractor was inserted to maintain visibility into the abdominal cavity.

An initial abdominal exploratory was performed, revealing approximately 20 mL of red, cloudy free fluid in the peritoneal cavity. Other findings included petechiation of the capsular surfaces of the left and right kidneys, an enlarged mottled spleen, an ectopic spleen approximately 1 cm by 1 cm by 1 cm in size, and edematous lymphatics of the liver. The urinary bladder was elevated with 3-0 Maxon stay sutures and isolated with lap sponges. Attached to the apex and ventral body of the bladder was friable, hemorrhagic omentum which, when removed, revealed a small hole in the bladder approximately 1 cm across. Surrounding the hole was a focal

spot of necrosis approximately 1 cm by 2 cm evident on the serosal surface of the bladder's apex. The necrotic tissue was excised from the bladder, allowing for a cystotomy to be performed. Smooth, yellow stones ranging from a few millimeters in size to 1 cm were removed with a bladder spoon. The stones were too numerous to count. Upon examination of the lumen of the bladder, multifocal necrosis was observed on the mucosa. The gray to black mucosa was debrided, revealing a viable purple submucosa.

A red rubber catheter was inserted into the urethra normograde, and warm sterile saline was flushed to remove many small stones and purulent debris from the urethra through the urethral orifice. An 8 French Foley catheter was passed into the urethra retrograde. The bladder wall was closed with two layers of suture: 3-0 Maxon in a simple continuous pattern for the first layer and a Cushing pattern for the second. Warm sterile saline was used to expand the bladder and check for leaks. No leaks were observed, and the saline was removed from the bladder. The abdomen was lavaged with approximately 2 L of warm sterile saline. A gauze and lap sponge count was performed prior to changing gloves and instruments. The abdomen was lavaged with another 1 L of warm sterile saline. The body wall was then closed using 3-0 Maxon in a simple continuous pattern. 4-0 Monocryl was used to close the subcutaneous tissue in a simple continuous pattern and the skin with an intradermal pattern. There were no surgical complications, and recovery from anesthesia was uneventful.

Ally was returned to ICU for monitoring. She was placed on Baytril (10 mg/kg IV q24h), carprofen (2.2 mg/kg SQ q12h), Cerenia (1 mg/kg IV q24h), methadone (0.1 mg/kg IV q4h), pantoprazole (1 mg/kg IV q 24h), Unasyn (30 mg/kg IV q8h), and LRS (36 ml/h or approximately 1.5 times maintenance IV). The bacteria cultured from Ally's urine and bladder mucosal biopsy were found to be sensitive to the antibiotics she was prescribed.

Urolithiasis can be treated medically or surgically. Aspects of both approaches were utilized to resolve Ally's urolithiasis. Antibiotic therapy and dietary modification are the hallmark components of medical management of infection-induced struvite urolithiasis.[40] Calcium phosphate stones, on the other hand, are less responsive to medical management.[60] However, due to the primary presence of magnesium ammonium phosphate in Ally's uroliths, the remainder of this discussion will be focused solely on the treatment of struvite urolithiasis. The following includes an in-depth look at medical management of struvite stones, as well as the surgical techniques utilized in Ally's case (cystotomy and partial cystectomy), with other techniques just briefly mentioned.

Antibiotic therapy should be based off of results of culture and sensitivity.[8] Amoxicillin, amoxicillin-clavulanic acid, and trimethoprim-sulfonamide may be used for empirical treatment while waiting for results. Clinical signs should begin to resolve within 48 hours of starting therapy. Treatment of simple cystitis or first time urinary tract infections is often done over 7 to 14 days. Complicated or recurrent infections may require 4 to 6 weeks of treatment. [8,24] Ideally, urine should be cultured prior to or immediately after termination of therapy to confirm resolution of infection. [8,57,61] Prognosis for urinary tract infection is good following appropriate therapy.[8]

In general, struvite uroliths can be medically dissolved unless surgical removal is indicated.[26] Common dissolution diets include Hill's Prescription Diet s/d and Royal Canin Veterinary Diet Urinary SO.[19,40] The primary goal of dissolution diets is to reduce urine pH and dietary magnesium.[56] These diets should not be fed for maintenance and should not be used in pregnant, lactating, or growing patients. Due to their high salt content, dissolution diets should also be avoided in dogs with congestive heart failure, hypertension, or nephrotic syndrome.

Dissolution diets may increase the risk of pancreatitis in Miniature Schnauzers due to a high fat content.[19]

Prevention of struvite urolithiasis is similar to medical management. Hill's Prescription Diet c/d and Royal Canin Veterinary Diet Urinary SO can both be used as maintenance foods.[40,61] Dogs should be provided fresh water at all times. High moisture foods may also be fed to increase water intake.[35] Monitoring and prevention of urinary tract infections is also important in the prevention of urolith formation.[26] Preventive measures are important, as recurrence of urolithiasis is as high as 25%. [15,34]

Uroliths that are nonclinical and unlikely to cause urinary obstruction do not have to be removed. On the other hand, stones that are associated with clinical signs or that are likely to cause urinary obstruction should be removed, preferably by minimally invasive procedures.[26] Urohydropropulsion, intra- and extracorporeal lithotripsy, transurethral cystoscopic stone removal, and minilaparotomy-assisted cystoscopic stone removal (percutaneous cystolithotomy) are some of the non-surgical options for removing uroliths from the urinary bladder and urethra.[3,29,61] While not appropriate for every case, these techniques should be considered over traditional surgical approaches whenever possible.[26]

The description of Ally's surgery above is an accurate representation of a routine cystotomy, minus resection of necrotic bladder wall, which was accomplished via partial cystectomy. Cystotomy is indicated for removal of calculi from the urinary bladder, as well as repair of bladder trauma, biopsy and culture of the bladder wall, biopsy or resection of bladder masses, ectopic ureter repair, and inspection or catheterization of the ureters in the case of idiopathic renal hematuria.[7,15,23,44]. Cystotomy for urolith removal is often considered an elective procedure for stones that are refractory to medical therapy or are associated with persistent

clinical signs; however, emergency cystotomy may need to be performed in the presence of bladder necrosis or if urinary tract obstruction cannot be resolved non-surgically.[3,6] In general, cystotomy is considered a safe and reliable method for removal of stones from the lower urinary tract. Nevertheless, post-operative radiography or ultrasonography should be performed to verify completeness of urolith removal, because upwards of 20% of cystotomy procedures fail to remove all calculi.[16]

Partial cystectomy is the removal of any portion of the bladder, either full thickness or partial thickness.[31] The main indications for partial cystectomy include bladder neoplasia and necrosis. Other reasons include patent urachus, urachal diverticula, and polypoid cystitis.[7,23,45,66] Surgical approach and isolation of the bladder is performed as described for a cystotomy. The bladder should be assessed for viability by looking at color, wall thickness, and blood supply, and all questionable tissue should be excised.[7] It is commonly reported that 75% of the urinary bladder can be excised as long as the trigone is preserved, with regeneration of normal size and function within 3 to 6 months.[6,23,45,66] Upwards of 90% of the bladder has been successfully removed in some cases of neoplasia and necrosis. In such cases of radical or total cystectomy, urinary diversion or bladder reconstruction techniques may be required to prevent urinary incontinence. [23,72] Urine can be diverted into the gastrointestinal tract, prepuce, or vagina, with each technique having different success rates and complications.[23] The bladder may also be reconstructed using autologous gastrointestinal tissue, porcine small intestinal submucosa, or any of a variety of synthetic or bioengineered autogenous graft materials.[23,70,71] The goal of bladder augmentation is to maintain bladder integrity, allow for urine storage and evacuation under low pressure, and preserve function of the upper urinary tract.[12,47,67]

Following bladder surgery, it is important to close the bladder wall with appropriate suture material and technique. A watertight closure can often be accomplished with a simple continuous or simple interrupted pattern. If leakage is still a concern, a second layer can be performed with an inverting pattern, such as Cushing or Lembert.[23] An absorbable monofilament suture material is preferred, ranging from 3-0 to 5-0, with a taper point needle.[6,15,23,44] Suture should engage the submucosa, which is the holding layer of the bladder and, ideally, not enter the mucosa in order to decrease risk of suture-associated urolith formation.[7,15,23,44] The bladder heals through a combination of epithelial regeneration and proliferation, synthesis and remodeling of scar tissue, and hypertrophy and proliferation of smooth muscle. Stretching of the bladder wall aids in return to normal lumen volume. [23,45] With appropriate anatomical alignment, bladder mucosal defects heal within 5 days, and the bladder regains normal tissue strength within 2 to 3 weeks.[6,23,44,66] In the case of debridement of the mucosal lining of the bladder lumen, as with Ally, the bladder often resurfaces completely within 30 days.[66]

It should be noted that in the event of urethral obstruction, urethral surgery is discouraged. Stones should be removed with minimally invasive techniques or should be pushed into the bladder and removed during cystotomy.[26] While controversial, cystocentesis may be used in some cases to relieve pressure in the bladder until obstruction can be resolved.[6] If detrusor atony occurs following prolonged outflow obstruction and bladder distension, the bladder should be kept empty for days to weeks. This can be accomplished by frequent bladder expression or an intermittent or indwelling urinary catheter.[10] Whether obstruction has resulted in urinary tract rupture or not, fluid therapy should be implemented to correct electrolyte and metabolic abnormalities and dehydration.[6,57] Diversion of urine is necessary to remove urine from the abdomen and prevent peritonitis.[6] In the event of septic peritonitis, surgical exploratory

and lavage should be performed. Patients often require 3 to 5 days of hospitalization to receive antibiotics, analgesia, fluid therapy, and, sometimes, nutritional support.[41,51] A favorable outcome is expected in patients that are eating and drinking on their own, afebrile, and comfortable 2 to 5 days following surgery.[51] However, despite therapy, mortality rates range from 20-68% if sepsis or septic shock occur.[38]

Case Outcome

On November 4, 2016, Ally's methadone was discontinued and replaced with tramadol (5 mg/kg PO q8h). She was transitioned to all oral medications on November 6, being placed on Baytril (10 mg/kg PO q24h), carprofen (2.2 mg/kg PO q12h), Clavamox (18 mg/kg PO q12h), and omeprazole (2 mg/kg PO q 24h), in addition to the aforementioned tramadol. Ally's Foley catheter was pulled on November 7, and she was transferred from ICU to the surgery wards. Ally urinated normally and was discharged on November 8, with Baytril, Clavamox, and tramadol. She was placed on Royal Canin Veterinary Diet Urinary SO. Her owners were instructed to return for an incision recheck in 2 weeks and for a urine culture 1 week after discontinuing antibiotics. On November 29, 2016, a call was made to check on Ally's status at home. Ally's owner reported that Ally had passed away in the weeks following discharge. An exact date was not provided, and necropsy was not performed.

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