Under Pressure

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Introduction

Obstructive urolithiasis is a common and costly urinary tract disease that occurs in ruminants. It is the most common cause of urinary tract disease in small ruminants.[7] There is no sex predilection; however, males are at an increased risk for obstructive urolithiasis due to the anatomical conformation of the urethra. The urethra of males is longer, it is curved due to the sigmoid flexure, and has a urethral process. The urethral process, also known as the vermiform apparatus, is an extension of the urethra at the distal end of the glands penis. The urethral process is narrowed in diameter when compared to proximal portions of the urethra.[5,6]

Uroliths can obstruct the urinary tract anywhere from the renal pelvis to the urethra. The most common sites for urinary tract outflow obstruction occur at the the distal sigmoid flexure and the urethral process. The urethra or urinary bladder can rupture as a consequence of obstructive urolithiasis at these sites.[7] Uroliths can be non-obstructive, can have a partial obstruction, or can completely obstruct urinary outflow.[4]

Clinical disease can vary depending on the site of obstruction, the severity of obstruction, and the duration of the disease. Partial obstructions can become complete as the duration of disease continues due to ongoing inflammation and damage of the urethral mucosa. Clinical signs related to obstructive urolithiasis in a goat range from inappetence to colic. Obstructed goats are commonly restless, are vocal, have stranguria, and frequently posture to urinate.[7] An obstructed goat requires emergency intervention since death is inevitable if medical and/or surgical correction is not obtained.[1]

History and Presentation

On June 27th, 2017, an approximately 6 months old whether named Remington presented to Mississippi State University College of Veterinary Medicine Large Animal Hospital due to stranguria and anorexia. He had a history of straining to urinate since June 24^{th}, which waxed and waned over the next three days. Remington was castrated two weeks prior to presentation. His diet consisted of hay and water ad lib and a commercial goat grain. Remington was diagnosed with struvite urolithiasis, which was treated with a routine tube cystostomy and recovered uneventfully.

On January 1^{nd}, 2018, Remington re-obstructed. His owner gave him ammonium chloride orally twice that day, but Remington's clinical signs of stranguria persisted. He became inappetent and depressed. Remington was examined by his primary veterinarian, abdominal radiographs and ultrasound were performed. No apparent radiopaque uroliths were observed on radiographs, but there was sludge evident in his bladder via ultrasound. Remington received tulathromycin (2.5 mg/kg) and flunixin meglumine (1.1 mg/kg). He was referred to Mississippi State College of Veterinary Medicine for further treatment. His main diet consisted of grass and hay. He was also supplemented with approximately one cup of Producers Pride DQ medicated sheep and goat feed one to two times a month.

On presentation to Mississippi State College of Veterinary Medicine, Remington weighed 14.5 kg. His temperature was 101˚F, his heart rate was 140 beats/min, and his respiratory rate was 32 breaths/min. He was quiet, alert, and responsive. He had a kyphosis posture, a tense caudal abdomen, and had a palpable enlarged firm bladder. There was a urolith within the tip of the urethra at the level of his glans penis measuring approximately 1x1 mm.

Diagnostic Approach/Considerations

On June 27^{th}, routine diagnostics and care for an obstructed urolithiasis case were performed. Remington was able to urinate on his own and was discharged on July 24^{th}, 2017. Upon presentation for his second episode of obstructive urolithiasis on January 2^{nd} 2018, an abdominal ultrasound was performed to assess the size of his bladder. His bladder measured approximately 9 cm, no free abdominal fluid was appreciable, and his kidneys appeared within normal limits. Remington was sedated with acepromazine and a urinary catheter was passed retrograde to the point of the urethral diverticulum. At that time, a small, spherical, white stone was removed from the urethral opening in the glans penis, but no significant quantity of urine was passed. A blood cell count had evidence of a stress leukogram and a chemistry panel revealed a moderate azotemia, a mild hypoalbuminemia, and a mild elevated creatinine kinase.

Remington underwent general anesthesia and a tube cystostomy was performed. The uroliths collected during surgery were submitted to the Minnesota Urolith Center for stone analysis. He was maintained on intravenous Lactated Ringer's Solution at 100 mL/kg/day over the next 24 hours. Creatinine levels measured at 1.80 mg/dL the day after his surgery. An ammonium chloride regimen was started at 100 mg/kg orally and he was given prophylactic ceftiofur hydrochloride (Excenel) at 2 mg/kg subcutaneously once and procaine penicillin at 44,000 units/kg. For post-operative analgesia, Remington remained on flunixin meglumine at 1.1 mg/kg intravenously q24h and he was started butorphanol at 0.025 mg/kg subcutaneously q4h. He was encouraged to drink oral fluids with electrolyte solution to increase diuresis, and his daily oral fluid intake was quantified.

On January 11th, Remington's Foley catheter was plugged to challenge him to urinate on his own. At first, he was dripping urine out of his prepuce comfortably, but as the day progressed he started to have episodes of stranguria as well as having bruxism. Two Abdominal ultrasounds were performed, one in the morning and one in the afternoon. His bladder was empty on the first ultrasound, but it had some retention of urine in the second. However, the urine present was mild and the bladder was not full. Due to Remington's discomfort level, it was decided to unplug his Foley catheter and re-challenge him at a later date.

On January 18th he was challenge to urinate on his own again. He remained comfortable throughout the day and urinated on his own. An Abdominal ultrasound revealed that his bladder was adequately emptying. On January 22nd the Foley catheter was removed. Remington maintained adequate diuresis and an appropriate appetite throughout the following days. On January 23rd, the stone analysis of the uroliths removed during surgery returned as 100% silicate. He was discharged on January 27th, 2018.

Pathophysiology

A urolith is a polycrystalline concretion in the urinary tract composed mainly of inorganic or organic crystalloids, with the remaining portion being non-crystalloid protein matrix that precipitate in supersaturated urine. Urolith formation begins with the initiation phase, where a central crystal nidus develops in the presence of calculogenic crystalloids and supersaturated urine.[4] The central crystal nidus can occur via homozygous nucleation or from heterozygous nucleation, where a nidus is composed of pure crystals surrounded by crystal ion aggregates, or the nidus is composed of foreign material, such as desquamated epithelial cells surrounded by crystal aggregates, respectively.[11] The subsequent growth phase around the crystal nidus depends on the continual presence of factors that contributed to the nucleation of the urolith.[4]

Urolithiasis has a multifactorial pathogenesis that includes factors such as diet, urine pH, and urine concentration.[3] Goats fed high grain diets, low roughage, and a diet with a low calcium to phosphorus ratio commonly develop magnesium ammonium phosphate (struvite) and calcium phosphate (apatite) uroliths, whereas those that consume legumes, such as alfalfa and clover are at risk for developing calcium carbonate uroliths.[3,7] Consumption of oxalate containing plants and diets high in calcium are thought to contribute to the development calcium oxalate uroliths.[6,7] Risk factors for silicate urolith formation are diets containing a high calcium to phosphorus ratio.[10] Likewise, animals grazing on plants and soils high in silica in the western United States and Canada are at risk for developing silicate urolithiasis. The pH of urine has a significant role in the formation of uroliths. Precipitation of struvite, apatite, and calcium carbonate crystals occur in alkaline urine, but silicate and calcium oxalate stones are not affected by urine pH.[2,7,] Water balance in the body affects the concentration and volume of urine. A decrease in water consumption reduces urine output and increases urine saturation, which contribute to a favorable environment for calculogenesis to occur. [7]

Treatment and Management

Prevention of urolithiasis is best and can be attained by providing a proper diet. Increased forage in ration increases the amount of saliva produced for adequate mastication, which increases the excretion of phosphorus via the saliva. Maintaining a diet that consist of a 2:1 calcium to phosphorus ratio with a decreased magnesium content, as well as avoidance of risk pastures and maintaining an adequate water supply decreases the likelihood of urolithiasis. The addition of dietary sodium chloride at 1%-4% of dry matter content is recommended to increase water consumption and promote urine dilution.[11]

Dietary grains must be kept low since they have a high phosphorus and magnesium content.[2] Vitamin A in the diet should be monitored for any deficiencies since low levels of vitamin A results in desquamation of bladder epithelial cells, which can then act as a nidus for urolith formation.[9] It is recommended to delay castration in small ruminants since urethral diameter has been associated with testosterone and early castration is strongly associated with obstructive urolithiasis. Delaying castration until 3 months of age revealed a 2.5 increase in diameter of the urethra at the level of the sigmoid flexure, and delaying castration until the age of five months revealed a 3.5 increase in urethral diameter. Prophylactic amputation of the urethral process has also been recommended. [2,7] When small ruminants are fed mainly forage with an occasional handful of grain and provided a carrot for a treat, most urinary calculi can be avoided.

Optimal treatment and management obstructive urolithiasis requires evaluating the systemic health of the patient and using a multimodal approach to facilitate therapy. This includes fluid therapy to correct electrolyte derangements and fluid deficits, preventing infections from occurring, providing analgesia and anti-inflammatory medications, maintaining a patent route for urine excretion, and acidifying urine. Anxiolytics may also be added to therapy to suppress the anxiety a goat may have due to the inability to urinate.[2,7]

The use of acepromazine at 0.05 to 0.1 mg/kg intravenously (IV) is thought to decrease urethral tone and relax the retractor penis muscle via the alpha-antagonistic mechanism. Acepromazine is useful in medical management for its anxiolytic properties in patients that cannot urinate. Consider the risk of its use in patients that are hypotensive or hypothermic. The use of diazepam at 0.1 mg/kg IV to decrease anxiety and provide urethral relaxation may also be used. The use of alpha-2 adrenergic agonist such as xylazine are not recommended prior to relief of the obstruction due to their promotion of diuresis.[7]

It is paramount to reduce urethral inflammation and to decrease the likelihood of a urethral stricture secondary to inflammation. Nonsteroidal anti-inflammatory drugs should be implemented into therapy once renal perfusion has been established. Broad spectrum antibiotics should be implemented into therapy for the prevention and treatment of infections that result from damaged urethral tissue. Beta lactams have a good spectrum of activity and concentrate in the bladder since they are excreted in the urine.[2,7]

Fluid therapy is essential in correcting fluid deficits and stabilizing electrolyte disturbances. The use of 0.9% normal saline is a good first choice, but the type of fluid should be determined based on biochemical derangements of the patient. The biochemical profile of an obstructed goat has a higher frequency of post renal azotemia, hyperkalemia, hyponatremia, hypochloremia, hypophosphatemia, and increased total carbon dioxide.[1] The levels of potassium can be used as an indicator to determine degree of therapy needed. Hyperkalemia is detrimental to cardiac function and can lead to a dangerous bradycardia. The use of dextrose at 2.5% or 5% solution and insulin may be used to drive potassium intracellularly. Cardiac contractility can be improved by adding 20 mL of 23% calcium borogluconate to a liter of fluids. Sodium bicarbonate can be used to decrease potassium levels and to correct metabolic acidosis, but should not be mixed with solutions containing calcium.

Retrograde insertion of a catheter to establish a patent route for urine excretion is not recommended due to risk of further damaging the urinary tract. Retropulse therapy via a catheter may result in further morbidity of the patient due to the risk of acquiring urethral trauma or causing over distension of the bladder.[7] Small ruminants have a urethral diverticulum about 0.5 cm deep in the caudal part of the urethra located at the level of the ischiatic arch.[8] Retrograde flushing the urethra would cause a urolith to temporarily lodge into the urethral diverticulum, where it would likely fall back into the urethra and cause the patient to become re-obstructed.[7] However, retrograde insertion of an angiogram catheter into the urinary bladder of healthy goats to evaluate urethra patency, as well as providing a route for urine excretion and the instilling of chemolysing agents has been successful.[8] The relief would be temporary with retrograde catheterization, and would eventually require surgical intervention.

Amputation of the urethral process can be done to provide urethral patency, but urination may still be difficult due to the inflammation at the proximal portions of the urethra. Other surgical procedures to establish urine flow are urethrotomy, perineal urethrostomy, bladder marsupialization, and cystotomy with tube cystostomy can be performed as surgical treatment for obstructive urolithiasis. Surgeries involving the urethra are palliative or salvage procedures, and are not recommended in valuable goats unless there is a urethral rupture or severe damage. A consequence of urethral surgery is re-obstruction within months due to stricture formation at the surgical site. Tube cystostomy is a curative surgical procedure and is the surgical treatment of choice for breeding goats and pet goats[7]. The cost for a tube cystostomy ranges from $1500-2000.[2]

A nonsurgical therapy that has been used in treatment of obstructive urolithiasis is the infusion of Walpole's solution, which contains a mixture of sodium acetate and glacial acetic acid. It has been reported that 80% of obstructions are temporarily relieved via this method, but re-obstruction occurred in 30% of patients discharged. An ultrasound guided cystocentesis is performed to drain urine, 50 mL of Walpole's solution is infused into the bladder, then the urine pH is checked two minutes post infusion. This procedure is repeated until the pH of 4.5 is achieved.[7]

Management post-surgical correction of obstruction is imperative in preventing re-obstructing from occurring. It includes maintaining an adequate diet previously discussed in the prevention of urolithiasis and maintaining a urine pH of 5.5-6.5.[2,7] Anionic salts such as ammonium chloride have been used to prevent urolithiasis because they reduce the pH of urine, increase water consumption, and enhance diuresis.[3] The dose of ammonium chloride is started with 50 mg/kg and increased until the desired pH is achieved, not to exceed a dose of 200 mg/kg a day.[2] Considerations must be made when using ammonium chloride since it decreases palatability of diet, which decreases owner compliance.[2] Ammonium chloride at 1%-2% has been used to alter the dietary cation difference (DCAD) of rations and induce metabolic acidosis.[11] A DCAD of 0 mEq/day has been shown to maintain a urine pH of 6.0 to 6.5 and may serve as a target for diet DCAD for the prevention of urolithiasis; however, chronic use results in the return of normal urinary pH due to acclimation of the kidneys.[3] Drawbacks from the metabolic acidosis achieved from the use of DCAD include decreased feed intake, weight loss, and bone loss from calcium resorption.[2,7]

Case Outcome

Upon Remington's discharge from Mississippi State College of Veterinary Medicine on January 27th 2018, his owner was instructed to contact the University of Tennessee Shelby county extension specialist to evaluate the property and analyze the grass for mineral composition, which Remington consumes as part of his diet. Remington's owner was contacted on October 1st, 2018, approximately 8 months after his discharge. His owner reports that Remington is doing great. Remington continues to enjoy electrolyte solution and is staying adequately hydrated. She stated that Remington's diet is appropriate and he loves getting carrots as treats.

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