In small ruminants especially in caprine breeds clinical obstructive urolithiasis is most frequently seen in young males that have been castrated at an extremely young age because of marketing to the pet market rather than the production market. The stones can be composed of phosphate and calcium salts: calcium phosphate (apatite), magnesium ammonium phosphate (struvite), calcium carbonate, calcium oxalate or silicate. Goats that are kept as pets are at an increased risk of obstructive urolithiasis; urolithiasis is usually secondary to the type of diet (genetic component?) and exposure to excessive grain allowing them to be extremely overweight as well.

**Etiology and Pathogenesis**

Obstructive urolithiasis is a well recognized, highly prevalent, and costly disease of both production and the pet small ruminant population. The rapid rise in popularity and push for urban farming has increased the pet caprine population, with this there has been an increase the prevalence of this disease.

The definition of obstructive urolithiasis is the inability to void urine normally because of calculi obstruction in any part of the urinary outflow tract, with the most common site of obstruction being the sigmoid flexure of the urethra. Calculus formations are due to multiple nutritional, physiologic, management and disease causes. Anatomically the urethral orifice of the sexually immature male or castrated male is significantly smaller due to a lack of testosterone causing maturation of the urethra; this making early castration before sexual maturity a significant risk factor for blockage. Also, if exogenous growth promoting steroids (estrogens) are used in male animals this too will lead to an overall decrease in urethra size.

The most common cause of calculus formation is supersaturation of the urine with excretory solutes, high concentrate diets typically fed to the pet caprine population and also from the lack of water intake. Factors further predisposing supersaturation of urine include increased insensible
water loss, urine stasis, increases in urine pH allowing precipitation of phosphate solute, decrease production and secretion of mucus from the bladder.

Calculus composition is directly related to the type of diet; for example silicate urolithiasis is common when the diet is composed of grass hay or cereal grass hays as the majority of the diet. Hay high in oxalates promotes oxalate stone formation. Diet composed of high calcium containing grains or high legume or clover hay promotes the formation of calcium carbonate stones. Cereal grains that are typically fed to production meat or dairy goats and may make up part of the diet for pet goats are high in phosphorus and lead to an imbalance or inversion of the calcium/phosphorus ratio; thus causing the formation of triple phosphate stones. Urolith formation can be further exacerbated if there is a significant amount of magnesium in the diet leading to magnesium ammonium phosphate stone formation. The formation of urinary calculi has been evaluated in multiple nutritional studies in various species and continues to be a significant cause of morbidity and mortality in production food animals as well as the ever growing pet small ruminant population. The role of nutrition and over conditioning of the pet small ruminant population has significantly increased the presence of urolithiasis in this population.

Once the process of calculus formation is initiated the growth of the calculus or of multiple calculi is through concretion. The increase in production of mucoproteins acts as the matrix for further production through concretion. The excretory dietary minerals combine with the matrix and form the stone. A nidus such as a smoldering cystitis or desquamation of bladder epithelial cells further provides a matrix for the solubilized excretory solutes to congregate around and form a calculus. Furthermore, obstruction may not necessarily be completely due to calculi, but from anatomical factors. Female small ruminants rarely suffer from obstructive urolithiasis even though all predisposing factors are present for formation of stones and the stones consistently form. Anatomically the urethra is much larger and shorter allowing for passage of the stones. In males or castrated males calculi become lodged within the sigmoid flexure, distal urethra, or within the urethral process. When male small ruminants are castrated at an extremely young age the penis does not mature normally lack of breakdown of the preputial skin attaching the urethral process back to the distal shaft of the penis, thus allowing the penis to be hypoplastic in these young male goats. Early castration of small ruminants allows for urinary calculi to be lodged
within the extra small urethral process. The hypoplastic tissue also is extremely difficult to identify normal anatomic structures while trying to correct an obstruction.

**Clinical Findings**

Examination of all body systems other than the urinary system is paramount because they may be completely normal; however, the affected animal may have signs of eminent death depending on the time elapsed from the initial obstruction. The severely affected animal will have signs of severe cardiovascular depression with severe bradycardia (secondary to hyperkalemia), muscular weakness (loss of electrolytes and azotemia) from ruptured bladder, and hyper to hypoventilation from uremic depression of the central respiratory centers within the brain. Thorough physical examination usually indicates the severity of the urethral obstruction before ancillary tests are performed.

The initial stages of obstructive urolithiasis, the affected small ruminant show extreme agitation, restlessness, tail twitching and abnormal posture. Most blocked animals will stand with both their front and hind legs stretched out. As the disease progresses vocalization will ensue with persistent posturing and dipping of their dorsum with minimal amounts of urine produced to complete anuria. Digital rectal palpation will elucidate consistent pulsation of the pelvic urethra. External palpation of the perineal urethra reveals distension and pulsation during straining. Rectal prolapse may be present in severe cases due to the severe abdominal press. If possible deep abdominal palpation may reveal a large bladder however, commonly the abdomen is severely taught precluding abdominal palpation. The examination of the prepuce may have evidence grit/crystals and bloody urine giving indication that the formation of uroliths has been present for a period of time.

A careful examination of the urethra will potentially isolate the site of the obstruction if present within the distal urethra or the urethral process. The urethral process is the most common site for blockage from sediment or small calculi due to the significantly smaller size of the urethra. Pain and or evidence of swelling may be identified with deep prepucial palpation of the more proximal urethra.
The consequences of a missed or delayed diagnosis of obstructive urolithiasis or a failure of proper institution of therapy may lead to rupture of the bladder, rupture of the urethra or lastly rupture with development of severe uremia and subsequent death. Uroabdomen can be a difficult diagnosis in the initial stages because of minimal accumulation of urine within the abdomen also the body condition and contour of the animal may make even late detection difficult. Ballottement of an abdominal fluid wave only occurs after 40-50ml/kg of urine has accumulated within the abdomen. Rupture of the bladder usually occurs within 24-48 hours post obstruction. The rupture occurs at the apex because of the Law of Laplace stating that the maximum tension on the wall is related to the overall radius of the bladder. The rupture relieves the pressure and discomfort of the affected animal, thus allowing the animal to resume a more normal attitude until the uremic syndrome supervenes. If the urethra ruptures rather than the bladder there is evidence of urine accumulation in the subcutaneous prepucial or perineal tissue.

Uremic syndrome develops secondary to loss of the normal ability to excrete organic solutes. This leads the affected goat to become anorexic, weak, and depressed. Significantly advanced cases result in the patient being in a moribund state and the potentially fatal if not treated aggressively.

**Ancillary Diagnostic Testing**

Common routine blood work consisting of a complete blood count (CBC) and serum biochemical analysis (SBA) are warranted in the suspected urolithiasis case. The CBC is typically within normal limits however, evidence of mild to moderate chronic nonregenerative (normocytic normochromic) anemia may be present along with evidence of an inflammatory leukogram consisting of increased percent band neutrophils, decreased segmented neutrophils, decreased neutrophil to lymphocyte ratio (1:1 instead of 1:2), and increased production of fibrinogen.

The SBA typically has elevations in the blood urea nitrogen and creatinine. The presence of azotemia may be prerenal, renal, or post renal. Patients suffering from urolithiasis may have all three causes of azotemia being part of the clinical disease. In evaluation of animals with clinical azotemia it is important to not speculate on the severity of azotemia leading to the definition of
the prognosis but the prognosis being based on how well the animal responds to the therapy for the azotemia. The azotemia can become quite severe in the ruptured case due to a larger amount of soft tissue for equilibration of the BUN and creatinine. The ruptured bladder or urethra results in more severe alterations of the blood electrolytes: hyperkalemia, hyponatremia, hypoglycemia, hyperphosphotemia, and hypochloremia; the presence of the hypochloremia lead to a hypochloremic metabolic alkalosis.

Urinalysis and sediment examination should be included as part of the initial examination of the patient. Marked crystalluria supports the diagnosis of obstructive urolithiasis. Further examination of the urine for color, clarity, specific gravity; the use of multistix for further evaluation assesses for the presence of ketones, occult blood, urobilirubinogen, pH, glucose, and bilirubin.

Transabdominal ultrasonography provides a safe noninvasive rapid means for evaluation of the urinary tract. Performing transabdominal ultrasonography can be accomplished using a 3.5–5.0 mHz curvilinear or linear probe can be used to evaluate the integrity of the bladder wall and to evaluate for the presence of sludge or stones within the bladder. Further examination should include evaluation of both kidneys to assess for presence of hydronephrosis and uroliths present within the kidney parenchyma or renal pelvises. Ultrasonography of the perineal urethra and the distal abdominal urethra can also be performed; unfortunately ultrasonographic location of the urolith within the distal urethra is quite difficult and rarely fruitful. Lastly the ultrasound can be used for detection of free abdominal fluid, cystocentesis, and abdominocentesis.

Radiography rarely reveals the cystic or urethral calculi unless they are radiopaque; if radiography is attempted the use of contrast radiography (iodinated solutions) will drastically increase the potential for diagnosis of obstructive urolithiasis.

**Medical Treatment**

If the animal is diagnosed with urolithiasis pursuant of medical therapy depends on the severity of the disease, the stage of the disease, the nature and extent of the uroliths, the intended long term use of the animal, the frequency of the disease, and most importantly the financial
constraints of the owner. Medical management can only be pursued in the affected animal if the bladder wall is intact. Unfortunately medical therapy often fails in over 50% of the affected animals leaving surgical correction, slaughter, or euthanasia as the only other options.

If there is evidence of sandy grit or calculi present within the urethral process surgical amputation of the process may allow the animal to urinate normally. The distal urethra can also be clogged with sediment and a small 5-8 French tomcat catheter or polypropylene catheter can be passed up the distal urethra and using hydropulsion with sterile saline solution mixed with KY jelly may provide alleviation of the blockage. Unfortunately the urethral diverticulum located at the level of the pelvic urethra prevents complete retrograde flushing and catheterization. The removal of the urethral process and flushing of the distal urethra are relatively easy and none invasive procedures that should be performed in all small ruminants suspected of having urolithiasis. When hydropulsion for removal of stones is unsuccessful other therapies must be pursued.

All of these procedures require exteriorization of the penis. Chemical sedation will help ease this process using diazepam (0.1–0.5 mg/kg) intravenously, acepromazine (0.05–0.1 mg/kg) intravenously. A lumbosacral epidural using 2% lidocaine (0.1–0.2 ml/kg, not to exceed 15mls) will provide temporary paralysis of the retractor penis muscle allowing manipulation of the penis.

The use of acetic acid (Walpole’s solution) with a pH of 4.5 has been used for medical management and dissolution of struvite crystals, stones, and bladder sludge. The use of this solution requires either and indwelling tube cystotomy catheter, a percutaneous poly propylene catheter, or multiple needle cystocentesis procedures. In one case series the use of acetic acid resolved the urinary obstruction in 80% of the affected animals.

Control and Prevention

Whether the patient has clinical evidence of urolithiasis or a prevention program is being instituted both strategies must focus on dietary modification and urine acidification. Ruminant urine is highly alkaline favoring formation of calculi. Acidification of urine can increase the
solubility of the uroliths composed of magnesium ammonium phosphate (struvite), calcium phosphate (apatite), and calcium carbonate and thereby inhibit precipitation in the urine. Urine acidification can be accomplished through the addition of ammonium chloride salt to the ration at a daily dose of 0.5% to 1% of the total dry matter intake, 2% of concentrate ration, or 200–500mg/kg/bw/day. The palatability of ammonium chloride is poor making free choice feeding difficult to maintain; most times it must be mixed with some form of concentrate feed remembering that this type of feed promotes calculogenesis or some form of a treat for consumption. Alternatively it can be mixed into a sugar solution (mixed with jello or syrup) and drenched orally or frozen in the freezer and fed as a treat. Continuous therapy with ammonium chloride can lead to refractoriness to urine acidification therefore current recommendations are to continue the drug for 3 consecutive weeks and then discontinue for 1 week and then start back on the medication. Regular monitoring of the urine pH is necessary for monitoring the effectiveness of the drug, with the ideal pH of the urine being <6.5 per day.

Dietary management is a key step for dissolution of calculi, prevention, and management of the disease by decreasing the amount of excreted solutes in the urine. Dietary modification needs to be correlated to the type of calculi present and also to the area of the country that the affected animal is located.