UNDERSTANDING COMMON DISORDERS OF SMALL RUMINANTS

Thomas Passler, DVM, PhD, DACVIM
passlth@auburn.edu

INTRODUCTION

Small ruminants enjoy a growing popularity in North America and are kept for a variety of purposes including production of meat, milk, and fiber, brush control, showing and competition in livestock shows, and as pet animals. More than 50% of goat farms in the US have fewer than 10 animals, and almost all US goat farms (99.7%) are considered small-scale operations with fewer than 500 animals.[1] Over 67% of goat operations have been in existence for less than 10 years[1] resulting in varying levels of knowledge among small ruminant owners. Many of the common diseases of small ruminants are caused by an underlying component of poor management, and hence can often be prevented by appropriate care, including housing, monitoring, and feeding. As compared to cattle, the requirements for energy availability and quality of forages are greater for small ruminants and this is reflected in high selectivity when grazing or browsing. Sheep are grazers and select higher quality portions of available roughage. In contrast, goats perform poorly on monoculture pastures, and if given a choice, prefer a diet of approximately 80% browse and 20% grasses.[2] As is true for many illnesses, proper nutrition and management can prevent the most common diseases of small ruminants, including obstructive urolithiasis and pregnancy toxemia, which is much easier and rewarding than their medical or surgical treatment.

KEYWORDS

Sheep, Goats, Urolithiasis, Pregnancy Toxemia

UROLITHIASIS

Etiology and Pathophysiology

Although the formation of uroliths occurs in both sexes, clinical signs are almost exclusively observed in male ruminants. The anatomy of the male urinary tract may lead to accumulation of calculi and subsequent obstruction. The most common sites of urethral obstruction in small ruminants are the urethral process followed by the sigmoid flexure. Urolithiasis should be considered a metabolic disease, and different risk factors underlie its development. The mineral composition of the calculi is dependent on geographic region and diet. Small ruminants

on high-grain diets such show animals, pets, and finishing lambs most commonly develop struvite uroliths (magnesium ammonium phosphate) or apatite uroliths (calcium phosphate).[3] Diets containing a large portion of legumes predispose to calcium carbonate uroliths, and silicate uroliths occur in animals foraging on silicaceous plants as present in the western United States.[3] Castration before puberty is considered a risk factor for the development of urolithiasis. Wethers castrated at 5 months of age have significantly larger penile and urethral diameters as compared to those castrated at either 2 weeks or 3 months.[4] Castration of small ruminants anticipated to be at risk for urolithiasis should therefore be performed as late as possible.

Diet and water consumption are important risk factor for the development of urolithiasis. Diets high in cereal grains contain large amounts of phosphorus, and their consumption provides much additional phosphorus that has to be excreted by the kidneys. In ruminants, another route for phosphorus excretion is through salivary secretion with subsequent fecal elimination; however, high grain diets (especially in pelleted forms) elicit little cud-chewing activity and salivation, adding to the need for renal excretion.[5] Reduced water intake and resulting urine concentration are also important in the pathophysiology of urolithiasis. The disease is more common in the hot and cold months, when water intake is inadequate to facilitate high renal excretion. A change of water sources to less palatable water (e.g. chlorinated city water) can promote decreased water intake with resulting urine concentration and urolithiasis.

Clinical Signs

Affected animals show signs of abdominal pain and tread, swish their tails and grind their teeth.[5] Depression, lethargy, and recumbency are frequently observed. Tachycardia, tachypnea, and rumen stasis are common. Because a mild rumen bloat is frequently present and other clinical signs are similar, grain overload and rumen acidosis should be considered as differential diagnoses. Although urine dribbling may still be present, many affected animals are anuric. Evidence of gritty precipitates on the preputial hairs suggests the etiology. On ultrasound, a distended bladder, thickened bladder wall, and abdominal fluid may be observed.[5] In small ruminants, the bladder is more common to rupture, but as in cattle, urethral rupture may also be observed. On blood sample analysis, a stress leukogram, increases in BUN and creatinine, hyponatremia, hypochloremia, and acidemia may be present.[6] The serum potassium concentration is frequently low to normal as result of inappetence but can also be elevated.[6]

Therapy and Prevention

Both, surgical and medical management is possible and should be considered according to financial constrains, chronicity of the illness, and severity of the obstruction. In acute cases in which urine dribbling is still observed, medical management may be sufficient, but animals must be hospitalized for close observation and the possibility for timely intervention. Medical therapy consists of fluids to correct dehydration and electrolyte imbalances, anti-inflammatory drugs (flunixin meglumine at 1.1 to 2.2 mg/kg IV), and urine acidification (ammonium chloride, 200 – 300 mg/kg PO). Amputation of the urethral process is frequently necessary, when urine dribbling is absent or inadequate. To extend the penis, sedation should be given (acepromazine hydrochloride, 0.05 to 0.1 mg/kg IV or IM or diazepam 0.1 mg/kg IV).[3] The urethral process should be dissected at an acute angle using a scalpel blade. Scissor may crimp the urethral stump, decreasing the diameter of the new opening.[7] Although amputation of the urethral process provides immediate relief, the long-term prognosis for continued urination is guarded, especially if management changes are not implemented.

Another treatment option for struvite urolithiasis is the use of Walpole's solution (pH 4.5) that is instilled into the bladder and dissolves struvite uroliths and crystals that form only at alkaline ph. Under general anesthesia an 18 gauge 4 inch needle is inserted into the bladder under ultrasound-guidance. Once the bladder is emptied, 30-50 mL of Walpole's solution is instilled and again removed. This can be repeated 3-4 times until an additional 30-50 mL of Walpole's solution is instilled and left in bladder. Urine flow may resume in 24-36 hours with normal voiding occurring in 3-5 days. Some animals may require a second cystocentesis in 2-3 days.[8]

Surgical options include the perineal urethrostomy, tube cystotomy, or bladder marsupialization.[7] Each option can provide effective urine flow and bladder emptying, but the perineal urethrostomy should be considered a salvage procedure, as urethral strictures develop commonly. Tube cystotomy and bladder marsupialization procedures require several days of post-surgical after care, and because urine scalding occurs frequently with bladder marsupializations owners should be aware of cost and complications with each procedure.

When an animal presents with clinical urolithiasis, the remaining herd members should be considered at risk, and management changes implemented. In the prevention of struvite uroliths, dietary management is most important. The reduction of grain for male ruminants or complete elimination of grain from pet goat diets is essential. Increases in roughage to grain ratios, feeding of lose rather than pelleted feeds, and correction of the

calcium to phosphorus ratio to 2:1 are useful. In feedlot animals or when owners chose not to reduce the grain intake, quantity and quality of water should be assessed. The addition of 5% NaCl to diets increases thirst and water intake. Ammonium chloride supplementation (0.5% - 1.0% dry matter) can be useful but reduces the palatability of feedstuffs and animals may sort out loose salts.

PREGNANCY TOXEMIA

Etiology and Pathophysiology

Pregnancy toxemia is among the most common metabolic disorder of sheep and goats, and is the result of a negative energy balance. Various risk factors exist but a decreasing plane of nutrition and resulting hypoglycemia and hyperketonemia are central to the pathophysiology. Pregnancy toxemia typically occurs in the last trimester (especially last month) of pregnancy. At that time, the energy demand of the growing fetus may exceed the energy availability. Ewes and does carrying multiple fetuses are at increased risk, as females carrying twins require 180%, and those with triplets 240% more energy than females carrying singletons.[12] Large or multiple fetuses inhibit the abdominal space, further limiting the amount of food intake. Primary pregnancy toxemia occurs when the plane of nutrition is decreased in late pregnancy, but other stressors and risk factors exist. Over-condition at the end of pregnancy is a risk factor, because excessive amounts of fatty acids are released from storage and reach the liver. The liver is unable to process the large amount of fat and the glucose production is further inhibited. Any stressors, including inclement weather, illness, and handling can tip the balance between subclinical and clinical negative energy balance. The physiological response to negative energy balance is the mobilization of fatty acids for energy production through the Krebs cycle, but at the same time, a high fetal demand for glucose exists. A conflict between gluconeogenesis and energy production develops as liver stores for oxaloacetate are diminished. As free fatty acids can only be incompletely removed from the liver, and energy demands persist, ketone bodies are produced. Although ketone bodies provide energy to many tissues, they further decrease appetite and glucose production.

Clinical signs

Clinical signs of those of a hypoglycemic encephalopathy. Affected animals are separated from the herd, depressed or somnolent. Tremors, opisthotonos, stumbling and falling can be observed in later stages. Signs of

abdominal discomfort, including grinding of teeth may be seen. Affected animals appear blind, walk into objects, and stand in abnormal postures, such as star-gazing. Intermittent convulsions can be observed and are followed by more normal periods.[12] Blood glucose concentrations are often low early in the disease but increase upon death of fetuses. Elevated ketone body concentrations in blood or urine are suggestive of the etiology.

Therapy and prevention

Depending on the stage of the disease, financial constraints, and whether dam or offspring are the primary interest, medical and/or surgical therapies are possible. The goal of therapy must be the discontinuation of the fetal energy demand, and a caesarean section is often the best choice for therapy. For surgical candidates, appropriate fluid therapy and glucose replacement should be chosen. The termination of pregnancy by injection of prostaglandins and/or steroids is suggested when the value of the patient does not allow surgery,[13] but may not induce parturition quickly enough. Administration of 50% dextrose (100 to 250 ml) temporally restores blood glucose concentrations and decreases the glucagon: insulin ratio, which inhibits further fat mobilization. Preferably, 5% dextrose solutions should be given as continued-rate infusions. Further supportive care includes the administration of rumen transfaunate from a healthy donor, high quality feeds, and oral propylene glycol (60 ml BID) as a ruminal propionate precursor.

The separation of dams into high and low risk groups aids in prevention as appropriate feeding plans can be designed for each group. To prevent secondary pregnancy toxemia, stressors such as transport or regrouping of animals should be avoided in late pregnancy. At-risk animals may be drenched with propylene glycol and/or receive ionophores which increase the availability of ruminal propionate for gluconeogenesis. Ionophores are beneficial for prevention but not treatment, because ruminal adaption to these antibiotics takes 3-5 days and results in temporarily decreased feed intake.

REFERENCES

[1]USDA APHIS NAHMS, **http://www.aphis.usda.gov/animal_health/nahms/smallscale/downloads/Small-scale_goat.**pdf, 2011; [accessed Jan 25, 2012]; [2]Rankins DL, et al. in: **Sheep and Goat Medicine**, Pugh DG, et al. 2012:18. [3]Jones M, et al. in **Sheep and Goat Medicine**, Pugh DG, et al. 2012:325. [4]Bani Ismail ZA, et al. **Bulg J Vet Med** 2007;10:29. [5]Van Metre DC in: **Large Animal Internal Medicine**, BP Smith. 2009:949. [6]George JW, et al. **J Am Vet Med Assoc** 2007;230:101. [7]Wolfe DF, et al., **Large Animal Urogenital Surgery**. 1998. [8]Janke JJ, et al. **J Am Vet Med Assoc** 2009;234:249. [9]Waller PJ, et al. **Vet Parasitol** 2004;122:207. [10]Miller JE, et al. in **Sheep and Goat Medicine**, Pugh DG, et al. 2012:106. [11]Fleming SA, et al. **J Vet Intern Med** 2006;20:435. [12]Edmondson MA, et al. in **Sheep and Goat Medicine**, Pugh DG, et al. 2012:150. [13]Brozos C, et al. **Vet Clin North Am Food Anim Pract** 2011;27:105.