Common Nutritional and Metabolic Diseases of Small Ruminants

By Stacy H. Tinkler, DVM, Dipl. ACVIM, Purdue University

Pregnancy toxemia ("twin-lamb/kid disease")

Overview

Pregnancy toxemia (PT) is one of many metabolic disorders that affect pregnant small ruminants. Its etiology has its foundation in the abnormal metabolism of carbohydrates and fats, which occurs at the final stage of pregnancy (Edmondson et al. 2012). More specifically, this disease is due to a lack of glucose as an energy source, either from poor nutrition, excessive demand from multiple fetuses, or a combination of the two. Recent evidence suggests that the glucose homoeostatic system of ewes bearing twins is significantly more susceptible to hypoglycemic stress than that of ewes bearing single lambs. These findings also show that the primary cause of hypoglycemia in late gestation twin-pregnant ewes is an increased susceptibility to a stress related reduction in glucose production rate rather than due to increased uptake by the fetus(es) (Schlumbohm and Harmeyer 2007). Any condition causing increased energy demand or decreased energy intake which results in a negative energy balance can be a cause. There are four categories of disease according to Edmondson et al. (2012) 1) primary PT caused by inadequate nutrition (poor quality feed, period of fasting) 2) fat ewe/doe PT seen in over-conditioned ewes/does in early gestation (suffer a nutritional decline in late gestation, possibly from smaller rumen capacity) 3) starvation PT seen in severely under-conditioned ewes/does (lack of feed after drought, heavy snow or flood) and 4) secondary PT due to concurrent disease such as parasites, poor dentition, or lameness. Animals carrying twins or triplets require 180% or 240% more energy, respectively, than those with a single fetus (Ermilio and Smith 2011).

Epidemiology:

The disease is seen more often in ewes than does, and animals in late gestation are at greatest risk (Van Saun 2000). Other important predisposing risk factors are either being excessively lean BCS (body condition score (<2/5) or obese, the presence of 2 or more fetuses, and some additional stressor (weather, sudden feed changes, disease, transportation). In severe outbreaks, morbidity can reach up to 20% with a mortality rate of 80% of affected animals (Ismail et al. 2008).

Clinical signs:

Clinical signs generally develop in 3-10 days and very few may be seen in the early stages. Signs are variable and can include: anorexia, depression, separation from the herd, neurologic signs (depression, tremors, star-gazing, ataxia, circling, teeth grinding, blindness) followed by recumbency and coma as the disease progresses. Other typical pertinent findings are
hypoglycemia and hyperketonemia/hyperketonuria of affected animals. The neurologic signs are thought to be due to cerebral hypoglycemia.

**Diagnosis**: urine or blood ketone levels (> 0.7 mmol/L), “acetone breath.” Urine ketone concentrations are more sensitive and specific than blood, (Edmondson et al 2012) and measuring urine ketones is the single most useful test but samples may be hard to collect in small ruminants. Urine ketone strips detect acetoacetate and acetone but not BHB, the primary ketone produced. False-negative or underestimation may occur depending on the ketone profile in the sample. Ketones are volatile and testing should occur immediately (Edmondson et al 2012). Elevated NEFA (non-esterified fatty acid), and BHB concentrations (>1.6 mmol/L) and low blood glucose concentrations can support the diagnosis.

**Differential diagnoses**: polio-encephalomalacia, peri-parturient hypocalcemia, toxic mastitis (if close to or after kidding), grain overload, listeriosis, lead poisoning.

**Treatment**

**Early stages (before recumbency)**: Treatment should be aimed at correcting any energy, electrolyte and acid-base disturbances as well as treating any dehydration and stimulating appetite. If the animal is still up and ambulating, a palatable, energy-dense, digestible feed should be offered and oral and IV glucose and electrolytes should be administered such as calcium 25 ml 23% calcium borogluconate/L, potassium (10-20 mEq/L) and 5% dextrose (Zamir et al 2009). Propylene glycol can also be administered as a glucose precursor (15-30 ml q 12 h), 30-60 ml propylene glycol PO q 12 h has also been recommended (Ermilio and Smith 2011). Rumen transfaunation and B-vitamin supplementation are recommended, particularly in anorectic patients.

When the ewe or doe is recumbent, treatment for PT must be initiated immediately and aggressively. Induction of parturition for removal of fetus(es) can be attempted in order to decrease energy requirements of the dam if the animal is at least at 135 d gestation (Radostits et al 2007); however, Brozos et al (2011) state that induction can be attempted in ewes after 140 d gestation and in does after 143 d gestation without compromising fetal development. Lambs or kids born more than 7 days premature don’t often survive (Edmondson et al 2012).

Various pharmacologic protocols have been proposed for induction of parturition in small ruminants [15 to 20mg dexamethasone (ewes/does), 10mg betamethasone (ewes), or 2.5 mg flumethazone (ewes/does)] (Brozos et al 2011). This protocol leads to parturition within 40 to 45 hours (ewes) or within 48 to 72 h (does). Simultaneous administration of 0.375 mg cloprostenol in ewes or 15 mg PGF2-alpha may enhance efficacy (Brozos et al 2011). 0.75 ug cloprostenol/45 kg BW in does (Erickson et al 2012) or 2.5-10 mg PGF2a (Edmondson et al 2012) has also been recommended.
It’s been suggested by Brozos et al (2011) that induction be followed by 20% dextrose solution (dose: 200–300 mL/animal, IV) or 50% dextrose solution (dose: 80–120 mL/animal, IV) twice daily until completion of parturition. Animals should be monitored regularly, as induction often leads to dystocia and retention of fetal membranes.

Flunixin meglumine (2.5 mg/kg BW, IM SID for 3 d) has recently been associated with significant improvement in survival rate of ewes and lambs born to affected ewes (Zamir et al 2009) as well as improved feed intake but should be used in conjunction with the aforementioned therapies. The mechanism of action is unknown.

Removal of fetus(es) should be performed by cesarean section in critical cases that are depressed or moribund (Edmondson et al 2012) and when the cost of surgery is warranted. Prognosis is generally poor in these cases; survival rate of c-section ewes and does did not exceed 60% and may be lower if the fetus(es) died in utero and were autolyzed. Therefore, euthanasia of affected pregnant ewes or does should always be considered before starting surgery. If surgery is elected, continuous fluids with dextrose is recommended (Edmondson et al 2012), (Brozos et al 2011).

After induced parturition or surgery, animals should be administered a broad-spectrum antibiotic, preferably injectable, an NSAID (eg, flunixin meglumine), and oxytocin (5 IU daily for 3 days) to facilitate expulsion of fetal membranes and to prevent metritis. The general condition of the ewe/doe usually improves after removal of the fetuses but this depends on the state of the fetuses at the time of removal.

Newborn lambs/kids from ewes/does with pregnancy toxemia are often premature with a suboptimal birth weight and are stressed. Massage and the administration of doxapram hydrochloride (5–10 mg/lamb/kid IV, SQ or SL) may be effective at stimulating respiration. If the dam cannot produce an adequate amount of colostrum, the lambs/kids should be given colostrum from another female in the flock/herd or from a “colostrum bank” at a dose of 50 mL/kg BW, 4 times in the first 24 hours of life. Supplemental feeding should be provided if the dam does not have enough milk.

Flock management/prevention

Pregnancy toxemia is a herd or flock health issue, and appropriate measures must be taken to prevent the disease in clinically healthy animals. Risk factors, both individual (age, poor dentition, lameness, or other disease) and flock/herd level (feeder space, protection from inclement weather or predators, poor quality forage) should be evaluated. Broad-spectrum anthelminthics could be considered if a diagnosis of GI parasitism is supported via quantitative fecal egg counts. Grouping pregnant animals by BCS and stage of pregnancy in order to help tailor feeding for physiologic state may be helpful. High-energy supplemental feed should be given to all pregnant animals on the farm such as 0.5-1 kg of cereal grain (corn, oats, barley or a combination) daily for ewes/does during the final months of gestation (Edmondson et al 2012). Free choice trace mineral salt mix should be available along with clean, palatable water. Dietary
niacin supplementation has also been recommended along with shearing sheep during late gestation.

The situation in the flock/herd should be reevaluated every 2 weeks. Animals at risk of developing PT can be identified for individual feeding to prevent development of the disease by measuring BHB concentration in the blood during the last month of pregnancy. Serum BHB concentration is useful for assessment of energy status in ewes. Values of 0.8-1.6 mmol/L suggest a negative energy balance and ewes or does with these values should be considered as having mild or subclinical PT (Ismail et al 2008). Animals found to have increased concentration of blood or urine BHB should be separated from other animals and monitored closely. If early signs of the disease are observed, individual treatment should be started immediately.

**Peri-parturient hypocalcemia (milk fever), “lambing/kidding sickness”, “downer ewe/doe syndrome”**

Hypocalcemia is an acute or subacute pathologic condition, which occurs shortly before or after parturition, although it is typically seen during the last 2 weeks of gestation (Edmondson et al 2012) when the fetal skeletons are mineralizing (Brozos et al 2011). Sheep experience hypocalcemia during late pregnancy as a result of insufficient dietary calcium to meet fetal needs. Insufficient calcium intake during late pregnancy or early lactation requires calcium mobilization from bones to meet this need.

**Epidemiology**

Disease incidence is associated with imbalanced nutrition and/or improper handling and housing and is usually less than 5%, but can occasionally be as high as 20%. Older animals are more susceptible due to an age-associated decrease in calcium absorption and mobilization. In dairy flocks/herds, the disease is more often seen after lambing/kidding, coinciding with peak milk production. Calcium concentration in ewes’ milk is almost twice that of cattle (Brozos et al 2011).

**Clinical signs**

Clinical signs include a temporary stiff gait, with muscle fasciculations which can progress to convulsions and occasional hyperesthesia or hyperactivity and hyperirritability have been reported. Animals become weak and less responsive, and become recumbent, with sternal progressing to lateral recumbency. Persistent calcium depletion leads to decreased muscle contraction and subsequent GI stasis and can lead to bloat in some cases and regurgitation and aspiration. Cold ears and skin are typical and rectal temperatures may be normal (Brozos et al 2011) or low. Poorly responsive, mydriatic pupils have been reported (Ermilio and Smith 2011).
Early stages: animals become isolated from the flock/herd. Subclinically affected animals are lethargic, have a poor appetite and are poor producers. The disease course can be hours to days, and may occasionally manifest as sudden death.

Later stages: depression and coma are often observed.

Diagnosis: is based on history and clinical signs but serum calcium concentration of $< 7 \text{ mg/dL}$ is seen in clinically apparent cases. (Edmondson et al 2012)

Hypocalcemia can often be seen as a secondary complicating factor with PT and ketosis (Brozos et al 2011). Differentiating between lambing sickness and PT is often difficult to do on the farm and definitive diagnosis requires blood calcium and BHB concentrations, and the two diseases can also co-exist.

Differential diagnoses include: polioencephalomalacia, advanced grain overload, toxic mastitis, lead poisoning and listeriosis.

Treatment

Uncomplicated hypocalcemia responds immediately (within 5 minutes) to IV calcium administration, and can be used to confirm the diagnosis. 30-60 mL of 23% calcium borogluconate IV usually is effective. Ermilio and Smith (2011) recommend a dose of 50-75 ml 23% calcium borogluconate per 45 kg BW. A solution containing phosphorus, magnesium, and/or potassium with dextrose can also be used. Administration must be performed slowly over 5 to 7 minutes, while heart rate and rhythm are monitored, and should be stopped at once if any heart rate or rhythm changes occur (Brozos et al 2011). Calcium administration can be repeated after 24 hours to avoid relapse. A total of 170 mL is the maximum recommended dose for a ewe or doe (Brozos et al 2011). If no response is evident, the diagnosis should be reevaluated as response to IV treatment is usually dramatic. Following IV or SQ injections, dietary calcium intake should be increased with alfalfa hay or calcium-based mineral supplements.

Prevention

No hard data exist as to the disease mechanism making it difficult to define specific feeding recommendations. Appropriate dietary calcium and avoidance of unnecessary stressors usually reduce the risk of disease in grazing animals (Brozos et al 2011). Cereal pastures and hays have low levels of calcium requiring supplementation; however, special attention should be given to feeds containing oxalates, which can form nonabsorbent compounds with calcium. The source of calcium is important as inorganic calcium is more digestible than calcium contained in feeds. Maintaining appropriate dietary calcium and phosphorus during late gestation to support fetal bone development is essential while being careful not to over supplement. Once into lactation, dietary calcium and phosphorus content should be increased to a level to support milk production
capacity. Cereal crop forages such as wheat or oat hay are very low in calcium as opposed to alfalfa hay and should be avoided unless the ration is balanced with other calcium sources.

**A word on hypomagnesemia…**

Magnesium deficit, leading to hypomagnesemia, is a disease of animals grazing young, rapidly growing pasture with reduced magnesium content, particularly during the spring or autumn (Brozos et al 2011). Grasses contain less magnesium than legumes and when growing rapidly in cooler conditions (lush spring pasture), magnesium availability is greatly reduced. The disease is rare in intensively raised ewes and does as they are fed concentrates to support increased production. The etiology influencing magnesium availability and absorption is multifactorial. The combination of low intake coupled with greater losses during early lactation result in the clinical syndrome.

**Epidemiology**

Increased potassium (eg, from alfalfa hay or haylage) and/or reduced sodium content in the diet, coupled with increased milk yield are the main risk factors for the disease. Adult ewes and does have a limited ability to mobilize magnesium body reserves and are dependent on daily intake to meet their needs. Stressors such as transportation can precipitate the disease.

**Clinical signs**

Clinical signs are caused by spontaneous activation of neurons in the central nervous system by the decreased magnesium concentrations in blood leading to tetany. Recumbent animals with seizures can die within hours.

**Treatment**

Administration of magnesium and calcium salts, separately or as a combination solution, is the recommended treatment when there is concurrent hypocalcemia. IV administration of a 4-5% magnesium chloride and 20% calcium borogluconate (50 mL) solution is ideal. Recovery is generally quick, but subsequent relapses are common; therefore, additional SQ administration, 12 to 24 hours later, is advisable. (Brozos et al 2011)

**Prevention**

Magnesium-rich mineral supplements should be provided before lambing and before grazing lush spring pastures. As most of these mineral supplements are not palatable, adequate feed consumption should be regularly monitored and ensured. When clinical cases occur in a flock/herd, it is advisable to provide clinically healthy animals on the farm with magnesium oxide at a dose of 7 g/animal PO. Mineral blocks containing magnesium and sodium chloride can help prevent sodium deficiency and promote magnesium intake.
Urolithiasis

Overview

Obstructive urolithiasis is the most common urinary tract disease of small ruminants. (Jones et al 2012). A 2001 NAHMS study reported that 20% of surveyed sheep operations have at least one case of urinary calculi in the previous 3 years. Urinary calculi (uroliths) are concretions of solid mineral and organic compounds that cause disease through direct trauma to the urinary tract and obstruction of urinary outflow. Urinary tract obstructions are primarily observed in males as a result of the length, narrow diameter, and tortuous nature of the penile urethra (Halland et al 2002). The urethral process is the most common site of obstruction in sheep and goats. In small ruminants that lack a urethral process (as a result of previous surgical removal or necrosis), the distal aspect of the sigmoid flexure is the usual site for blockage. There are a variety of surgical treatment options for urethral obstruction; however, recurrent postoperative urethral obstruction often limits long-term survival and quality of postoperative life. Other complications associated with treatment are common and include urethral stricture, cystitis, and incomplete clearance of calculi (Halland et al 2002). Therefore, prevention is paramount, and owners need to be made aware of the commitment to dietary changes that is needed in order to reduce the concentration of compounds in the urine that lead to the formation of urinary calculi.

Epidemiology

Risk factors for the development of urolithiasis include dietary imbalances, water restrictions, and urine pH (Halland et al 2002). Approximately 75% of uroliths found at Purdue University are struvite, with occasional calcium carbonate and silica stones (J Hawkins, personal communication). Calcium carbonate stones are associated with calcium and oxalate-rich forages that are low in phosphorus and magnesium (Halland et al 2002). Silica urolithiasis develops when animals are fed forage with high silicone content (rangeland grasses) in combination with deficiencies of copper and zinc, and struvite calculi are associated with grain-based diets containing high concentrations of phosphorus relative to calcium (Halland et al 2002). Pelleted rations may also predispose to the formation of phosphate calculi (struvite or apatite) by reducing salivary excretion of phosphorous. Acidic urine (pH < 7.0) favors the formation of silicate stones, whereas alkaline urine (pH > 7.0) favors the formation of apatite, calcium carbonate, and struvite stones. Radiopaque calculi (calcium carbonate, calcium oxalate, silica) do not dissolve readily by urinary acidification from addition of ammonium chloride to the diet or infusion of acidic agents into the bladder through a tube cystotomy ((Kinsley et al 2013).

Clinical signs

Clinical signs are variable and include depression or restlessness (treading, stretching, tail-swishing), vocalization during urination, bruxism, stranguria, urine dribbling, and anuria if completely obstructed. Look for urine crystals on the prepuce, and any ventral swelling (urethral/bladder rupture). Rectal prolapse and abdominal distension can also occur. Small
ruminants with serum K+ < 5.2 mEq/L and with no fluid in the abdomen at admission were significantly more likely to survive (Van Metre 2006).

**Diagnosis**

Diagnosis is often based on history and clinical signs alone. Deep abdominal palpation for an enlarged bladder and/or digital rectal examination to feel for urethral pulsation are also suggestive and helpful diagnostic aids. Trans-abdominal ultrasound in the right flank (caudal abdomen) to look for an enlarged bladder (> 6cm with a tight, round appearance) or abdominal radiographs can also provide more definitive information about the number and location of stones particularly if uroliths are radio-opaque (Kinsley et al 2013).

**Treatment**

Medical therapy is generally unsuccessful in these cases. Ultrasound-guided cystocentesis and percutaneous infusion of Walpole’s solution (pH 4.5) in male goats has had some success in dissolving struvite calculi but recurrence of urethral obstruction was fairly common (6/20 goats that were discharged (Janke et al 2009). This technique might be useful in situations where short-term resolution is the goal, as it is non-invasive, inexpensive, resolved obstruction in 14/17 goats, and doesn’t require use of drugs with long withdrawal times (Janke et al 2009). The most common complication with this technique is Walpole’s solution leakage from bladder puncture sites which could result in abdominal adhesions, and the acidity of the solution causes mucosal irritation and results in cystitis (Janke et al 2009). Percutaneous, self-containing urinary catheters, and cystocentesis provide short-term relief (24-36 h) prior to relieving urethral obstruction (Van Meter 2006). Laser lithotripsy coupled with urethroscopy has been reported (Halland et al 2002) and was used successfully to disrupt uroliths firmly lodged in the distal urethra but not freely movable liths as this precluded appropriate positioning of the laser and may be a worthwhile therapeutic option in some cases.

The owners’ goals (ie breeding) and financial resources are what determine the form of treatment. Surgical success depends in large part on duration of disease, and correction of fluid and electrolytes before or during surgery. Methods developed to reestablish urethral patency include perineal urethrostomy, penile amputation, urethrotomy, normograde and retrograde urethral flushing, cystotomy, and tube cystotomy (Halland et al 2002) and some of the advantages and disadvantages are summarized in the table below.
<table>
<thead>
<tr>
<th>Surgical procedure</th>
<th>Pros</th>
<th>Cons</th>
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</thead>
<tbody>
<tr>
<td>Vermiform appendage (urethral process) amputation</td>
<td>Fast, Easy, Immediate resolution urethral patency ~50%</td>
<td>Recurrent obstruction, Temporary fix (most reobstruct in &lt; 36 h)</td>
</tr>
<tr>
<td>Perineal urethrostomy</td>
<td>Sedation + local anesthesia, Epidural anesthesia</td>
<td>Salvage procedure, Stricture formation +/- recurrent obstruction (w/in 1 y post-op)</td>
</tr>
<tr>
<td>Tube cystotomy</td>
<td>76-80% success rate, &lt;20% recurrent obstruction, Can perform in field</td>
<td>$, general anesthesia, Catheter problems, Adhesion formation, 2 wk hospitalization</td>
</tr>
<tr>
<td>Bladder marsupialization</td>
<td>Salvage procedure, Consider if client will not be compliant with dietary changes?</td>
<td>$ (but cheaper than tube cystotomy), general anesthesia, Urine scald, UTI, Stricture, Bladder mucosa prolapse</td>
</tr>
<tr>
<td>Cystotomy with urethral flushing</td>
<td>Shorter duration of hospitalization than tube cystotomy</td>
<td>$, general anesthesia, Laparotomy, May increase risk of urethral rupture</td>
</tr>
<tr>
<td>Urethroscopy + lithotripsy</td>
<td>Less invasive, Shorter hospitalization, Less expensive than sx</td>
<td>Only works on stones in distal urethra</td>
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Currently, tube cystotomy appears to be the most appropriate approach for obstructive urolithiasis in small ruminants for use as breeding animals or pets, with success rates ranging from 76-90% in the short-term and 86% long-term (Ewoldt et al 2008).

**Prevention**

Because of the poor prognosis and cost of clinical cases of obstructive urolithiasis and the implications for the herd/flock, prevention must be a focus. Risk factors to address in preventative strategies include high dietary phosphorus/calcium ratio, high dietary magnesium, low fiber content of rations, low urine output, and an alkaline urine pH (Ewoldt et al 2008). High phosphorus levels are prevalent in grains (sorghum, wheat, corn, milo, and oats in particular), and the Ca:P ratio should be 2:1 or 2.5:1. Phosphorus excretion may be minimized by feeding more roughage and less pelleted ration, as this will also enhance salivary excretion of phosphorus. Elimination of alfalfa feeding and reduction of grain feeding with a change to grass hay as primary forage, along with increased grazing and anion salt supplementation have also been recommended (Van Metre 2006). Reduction in alfalfa and grain reduce the calculogenic
load (Ca and P). Increased water intake and urine volume is an important preventive measure as well. Palatable water sources are essential, and water intake is increased in animals on forage vs pelleted diets.

Urine pH is also an important factor in urolithiasis, with a urine pH recommended goal of 5.5-6.5 for prevention. Anionic salts containing mainly chlorides (sodium chloride 1-4% DM, calcium chloride and ammonium chloride 0.5-2% DM) have been used extensively in order to reduce urine pH, increase urine output and subsequently help prevent urolithiasis but have had inconsistent results. This is most likely due to the addition of these salts to the diet as a simple percentage without considering the total ration (Navarre et al 2012), or may be due to the multifactorial nature of urolith production and high-potassium diets that interfere with urine acidification (Jones et al 2009). Additionally, it appears that there is renal adaptation to the ammonium chloride and long-term acidification is not maintained (Jones et al 2009). Urine pH must be monitored in animals consuming diets designed to prevent urolithiasis. Some negative effects of anion administration include reduced palatability, and bone loss when ingested long-term (Jones et al 2009). It has been suggested that a target DCAD of 0 mg/kg may serve for diet formulation for urolithiasis prevention (Jones et al 2009), as this DCAD resulted in a decreased urine pH (6-6.5) after 5 days of feeding with no change in blood pH (no acidosis) that could lead to bone loss. Struvite crystallization occurs only at a pH range of 7.2 to 8.8, and dissolution occurs at pH < 6.5. Apatite uroliths develop at a urine pH of 6.6 to 7.8 (Jones et al 2009).

Delaying castration to at least 3 m, 4-6 will decrease the risk for urinary tract obstruction. Urethral diameter is significantly larger (3.5x) if males are castrated after 5 mo. If castrated after 3 mo, diameter is 2.5x larger. This is particularly true in lambs. (Angelos 2013)

**Rumen acidosis (“grain overload”)**

**Overview**

Rapidly fermentable sugar or starch is consumed in excess and lactic acid is overproduced, accumulates, and leads to a severe decline in rumen pH. As the rumen pH declines, Lactobacillus bacteria will start to proliferate generating more lactic acid. Lactic acid production increases rumen osmotic pressure and fluid is drawn into the rumen from the systemic circulation leading to dehydration, and in severe cases, hypovolemic shock (Navarre et al 2012). Lactic acid is irritating to the rumen, and bacteria and toxins can access the systemic circulation through damaged rumen epithelium. Lactate concentrations increase in the blood leading to systemic metabolic acidosis.

**Epidemiology**

Corn is commonly implicated; however, other cereal grains, especially if finely ground, may also cause this disease. Bread, candy, apples, fruits, beets, and potatoes have been implicated as well. Rumen acidosis (RA) usually occurs in animals on a primarily forage-based ration which
suddenly have access to large amounts of readily fermentable concentrates (Navarre et al 2012) or in concentrate-fed animals where the amount was suddenly and significantly increased. For example, in cases of extreme weather changes or varied water availability or if ration mixing errors have occurred such as rumen buffers like monensin being left out of the ration.

**Clinical signs**

Signs first appear 12-36 h post-ingestion and are variable depending on the amount and type of feed. (Navarre et al 2012) Clinical signs range from anorexia, depression and weakness to recumbency in cases of severe shock. A fluid-filled rumen can be ballotted and/or ausculted. Diarrhea and acute laminitis have also been reported. In chronic cases, laminitis and foot abscesses can be seen. Acutely acidotic goats may show neurologic signs such as ataxia, head-pressing, opisthotonos, seizures, recumbency, cold extremities and dilated pupils. Secondary thiamine deficiency may be a contributor to neurologic signs (Navarre et al 2012).

**Diagnosis**

History and clinical signs, coupled with rumen pH and examination of rumen fluid are needed for definitive diagnosis. Fluid is milky gray, and pH may be < 5.5. Rumen protozoa are usually decreased or absent and large gram negative rods may be seen. Lab data are consistent with dehydration and metabolic acidosis. Liver enzymes may be elevated on, and the leukogram is Ismail variable (normal to degenerative left shift) depending disease severity.

**Treatment**

Treatment is aimed at correcting shock, dehydration, acidosis, toxemia and removal of feed. IV sodium bicarbonate (5%) should be administered. NSAIDs (flunixin meglumine 1.1-2.2 mg/kg IV BID) are indicated for pain and inflammation. In mild cases, reduction in grain feeding along with supportive care is usually sufficient. Supportive therapy includes intravenous and oral fluids, rumen transfaunation, alkalinizing solutions PO or IV, and antibiotics (procaine penicillin G 22,000 U/kg IM BID). In more severe cases intensive supportive care and rumenotomy may be indicated. Once rumen pH is corrected, rumen transfaunation is needed (1 quart fluid). Thiamine supplementation (10 mg/kg IV or SQ TID-QID) is also recommended. Secondary liver abscess and/or fungal rumenitis are potential sequelae that need to be considered but liver abscesses occur less often in sheep and goats than in cattle (Navarre et al 2012).

**Prevention**

Any feed changes need to be made gradually over several weeks so the flora have time to adapt. Grains should never be fed prior to a forage meal as forage consumption initiates salivary buffering. Another recommendation is to divide the grain ration into 3 or more separate feedings depending upon the amount needed to support production level. Animals on high-grain rations could have buffering agents added to the diet. Sodium bicarbonate can also be offered free-
choice as part of the formulated ration. Calcium carbonate (limestone) and magnesium oxide are other buffers that can be used.
References


