Dinner plan killers: Blocked goats, pig C-sections, and pregnancy toxemia

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Abstract

Urinary obstructed small ruminants, pigs in dystocia, and pregnancy toxemia all represent challenging, truly emergent clinical scenarios in mixed and large animal practice. Each of these scenarios, however, lend themselves to protocol development. Putting these cases on autopilot can remove a lot of the associated stress and ensure consistent patient care with consideration for client expectations and finances built right in.

Initial evaluation of animals with suspected urolithiasis should include examination of the preputial hairs for grit or formed stones and exteriorization of the penis for examination and amputation of the vermiform appendage. When vermiform appendage amputation fails to relieve the obstruction, catheterization and surgical intervention, including perineal urethrostomy, tube cystotomy, minimally invasive tube cystotomy, and others should be considered.

Swine presented for dystocia are often compromised and require a Cesarean section. Having a standard protocol in place for perioperative drugs, including anesthetic drugs, antimicrobials, and anti-inflammatories, reduces the veterinarian's stress in preparing to go to surgery. Unlike other species, the surgical approach in swine is paramammary and the uterus is much more highly vascularized. Care must be taken to minimize abdominal contamination where dead piglets are involved.

Ewes and does presented ill in late gestation should always be evaluated for pregnancy toxemia. Many, if not most of these animals, additionally have pneumonia and a GI parasite burden. Does and ewes that are still standing and eating can often be managed in the field, while those that are recumbent will only recover if placed on IV fluids and dextrose CRI. Producers must be educated on pregnancy toxemia risk factors, such as obesity, parasitism, and improper feeding.

Key words: urolithiasis, sheep, goats, swine, Cesarean section, pregnancy toxemia

Blocked Goats

Obstructive urolithiasis is considered to be the most economically significant urinary tract disease of food animals, affecting primarily intact and castrated male ruminants, swine, and camelids. Male small ruminants are particularly predisposed, while females are rarely clinically affected. Pathogenesis

Uroliths are solid crystalline formations in the urine which are composed of organic matrix and organic and inorganic crystalloids. Matrix, made up of sugars, proteins, and cells, results from urine super-saturation. Factors affecting urine super-saturation include the rate of renal excretion of crystalloids, negative water balance, urine pH, and the presence or absence of crystallization inhibitors. Metaplasia of uroepithelium, as a result of vitamin A deficiency, may contribute cells and protein for nuclear formation. Suture, tissue debris, blood clots or bacteria may also serve as nuclear components initiating urolith formation. Infection, however, is considered to be a minor factor in urolith formation in ruminants. The formation of a nucleus is followed by deposition of inorganic minerals, including magnesium, calcium and phosphate, onto the matrix.

The anatomy of the distal urinary tract of male ruminants differs significantly from that of males of other species, and contributes to the development of obstruction and increases treatment difficulty. The penis is sigmoid in arrangement, with 2 major bends occurring between the urinary bladder and the distal glans penis. The distal flexure is a common site of urethral obstruction by uroliths. The glans penis of the small ruminant also has a vermiform appendage, or urethral process, which is an extension of the urethra 2 to 4 cm beyond the distal end of the penis. It has a narrowed diameter compared to the more proximal portions of the urethra and also serves as a common location for obstruction. The urethral diverticulum, an outpouching of the urethra at the level of the ischial arch, complicates treatment of affected animals. When a urinary catheter is passed into the urethra in a retrograde manner from the glans in order to access the urinary bladder, this diverticulum readily accepts the catheter, rather than allowing the catheter to proceed into the urinary bladder.

Urolithiasis is a multifactorial disease with such inputs as diet, urine pH, and body water balance. Struvite (magnesium ammonium phosphate) and apatite (calcium phosphate) may be commonly seen in animals fed high-grain diets, while animals consuming legumes are predisposed to calcium carbonate uroliths. Silicate stones may be observed in animals grazing silicaceous plants and soils in the western United States and Canada. Calcium oxalate stones may be associated with oxalate-containing plants.

A significant factor in availability of urolith components and their binding ability is urine pH. Struvite, apatite, and

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calcium carbonate uroliths are known to precipitate in alkaline urine. Struvite crystallization occurs only at a pH range of 7.2 to 8.4, while apatite stones develop at a urine pH of 6.5 to 7.5. Calcium carbonate stones also tend to form in alkaline urine, while pH may have little or no effect on silicate or calcium oxalate uroliths.

Total body water balance plays an important role in calculogenesis by its effects on urine volume and concentration. This may be seen in winter and during times of other systemic illness, when animals consume decreased volumes of water. A negative body water balance contributes to supersaturation, precipitation, and formation of residue of organic and inorganic crystalloids in urine.

Uroliths may obstruct urine flow anywhere from the renal pelvis to the distal urethra, although the most common sites of obstruction are at the distal sigmoid flexure or the vermiform appendage in sheep and goats. Obstruction at these sites may result in either rupture of the urethra or of the urinary bladder.

Treatment

Management of affected individuals consists of establishing a patent route of urine excretion, providing analgesia, correcting fluid deficits and correcting electrolyte derangements, decreasing inflammation and preventing infection.

The presence of the urethral diverticulum prevents passage of a urinary catheter retrograde from the urethral orifice to the urinary bladder. Retrograde catheterization or retropulsion of uroliths is not recommended to avoid further trauma or puncture of the urethra at the level of the diverticulum. Attempts at retropulsion of uroliths may result in over-distention of the urinary bladder as the stone is diverted into the diverticulum, allowing fluid to pass into the bladder, followed by the urolith falling back into the urethra and preventing the bladder from emptying. Occasionally, removal of the vermiform appendage in small ruminants establishes a patent urethra; however, inflammation in the proximal urethra from passage of the uroliths may still prevent normal urination. Uroliths tend to occur in multitudes in the urinary bladder, and 80% of animals initially relieved by amputation of the vermiform appendage will reobstruct with subsequent stone passage. Relief of urinary obstruction most often requires surgical intervention.

Sedatives may be useful to facilitate treatment. Historically, acepromazine (0.05 to 0.1 mg/kg, IV or IM) has been utilized in the medical management of urolithiasis. Unproven arguments for utilization of acepromazine have been to relax urethral tone through α -antagonistic effects on smooth muscle and relaxation of the retractor penis muscle. Benefits of acepromazine may also include suppressing the anxiety associated with the inability to urinate. Caution should be taken when using phenothiazine tranquilizers in patients which may already be hypotensive and hypothermic. Diazepam (0.1 mg/kg, slow IV) may also be used for urethral relaxation and as an anxietolytic. Xylazine (0.05 to 0.1 mg/

kg, IV or IM) or other α -2 agonists may be used in attempt to restrain the patient for examination of the penis and have excellent analgesic properties in ruminants. Caution should be exercised when utilizing xylazine prior to relief of the obstruction, as it promotes diuresis, as well as enhancing hypotension. Lumbosacral epidurals using 2% lidocaine (1 mL/7 kg) may be utilized in the place of sedation to relieve discomfort and aid in exteriorization of the penis.

Fluid therapy should be instituted as indicated by the clinical findings and economics of the case. After relief of the obstruction, diuresis is important to replace dehydration, reduce azotemia, and flush the urinary tract. 0.9% NaCl is a good choice for intravenous fluid therapy, although additional electrolyte and acid-base abnormalities should be considered.

Non-steroidal anti-inflammatory drugs should be administered to decrease inflammation and aid in the prevention of urethral stricture formation, but should be used with caution until adequate renal perfusion is attained. Broadspectrum antibiotic therapy should be instituted to prevent or treat infection resulting from devitalized or inflamed urinary tissues or cavitational accumulation of urine. Beta-lactams (penicillins and cephalosporins) may be chosen, as they have good spectrum of activity and are excreted in the urine.

Many surgical options are available, usually selected based upon economic constraints and purposes of the animal. Animals with ruptured urethras should be considered lost for breeding purposes due to adhesion formation. Salvage procedures include perineal urethrostomy and penile amputation, both of which typically suffer from stricture formation weeks to months after surgery. Tube cystotomy is the most successful method of urine diversion, where a foley catheter is placed in the urinary bladder, passes through the abdominal wall and drains externally. This diversion allows for urethral healing and stone dissolution through flushing or diet change, and tubes are left in place until urine flow is achieved through the urethra. Other options include urinary bladder marsupialization, urethrotomy, and laser lithotripsy. Each of these methods is described in most food animal surgery texts.

Once the obstruction is relieved, treatments to acidify the urine should be initiated in an effort to solubilize additional stones and sediment. Ammonium chloride at a dosage of 90 mg/lb (200 mg/kg) may be orally administered initially and adjusted to attain a urine pH of 6.0 to 6.5. Care should be taken in dosing so that systemic over-acidification does not occur.

Prevention

Due to the poor prognosis and expense associated with clinical cases of obstructive urolithiasis as well as the herd or flock implications of the disease, considerable focus should be placed on prevention. The important role of metabolic by-products and minerals in the pathophysiology causes diet to be the primary focus of disease prevention. Risk factors addressed in preventative strategies include high dietary

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phosphorus relative to calcium, high magnesium, low fiber content of rations, low urine output, and an alkaline urine pH. Additional factors including selective grazing and castration timing may be addressed.

An elevated level of phosphorus in the diet, with a calcium:phosphorus ratio less than 2:1 increases the excretion of phosphorus in the urine and provides an ion to bind to organic matrix. Increasing the level of calcium in the diet markedly decreases the incidence of urolithiasis, probably due to competition with phosphorus for intestinal absorption and matrix binding. Phosphorus should not comprise greater than 0.6% of the total ration and it is recommended that a 2.5:1 or 2:1 calcium:phosphorus ratio be achieved, by the use of calcium salts, if necessary. Calcium oversupplementation should be avoided as increased calcium excretion in the urine may contribute to calcium-containing uroliths. High phosphorus levels are present in grains, particularly sorghum, wheat, corn, milo, and oats.

A reduction in phosphorus excretion into the urine is also desirable. Ruminants excrete phosphorus primarily by saliva, where it is then swallowed and removed from the body in the feces. Excessive dietary levels of phosphorus may saturate this salivary pathway, causing the excess to be excreted in the urine. Urine phosphorus excretion is greater in animals fed pelleted rations as compared to meal-type rations, due to a decrease in saliva production, and therefore a pathway for excess phosphorus excretion. Increases in the roughage component of diets are important from this standpoint, as they increase the amount of saliva that must be produced for proper mastication.

Particularly in the case of struvite stones, but also with apatite stones, an increase in magnesium excretion into the urine is contributory to crystallization. It is recommended that magnesium not exceed 0.6% of the total ration of ruminants. Magnesium is more available and absorbed more efficiently from concentrate rations than from roughage diets.

Increasing water intake and urine volume is an important preventive measure for urolithiasis. Sources recommend the provision of adequate palatable water at desirable temperatures according to the ambient environment. Ruminants demonstrate a reduction in water intake for grain feeding over roughage feeding. Additionally, the feeding of intermittent meals may cause shunting of body water into the rumen due to increased osmotic pull from generated volatile fatty acids, resulting in a decrease in urine output. This has led to the recommendation that ruminants be fed *ad libitum* to maintain urine output.

Increasing forage vs grain in the diet of animals at risk for urolithiasis has many benefits. Grain results in increased magnesium, phosphorus and peptides in the urine. Forage encourages saliva production for phosphorus excretion, potentially reduces magnesium uptake, reduces overall grain consumption and increases water intake. Legumes and their hay should be avoided, as they have high levels of calcium and are associated with calcium carbonate urolithiasis. The role of urine pH in urolithiasis is well documented and various sources recommend urine pH goals of 5.5 to 6.5, based on the solubilities of the common stone compositions. Due to an ability to alter acid-base balance and body water balance, salts have been widely used and recommended for the prevention of urolithiasis. Anionic salts containing primarily chlorides have been popular and used extensively, as they reduce urine pH, increase urine output, and, ultimately prevent urolithiasis. Sodium chloride (1 to 4%), calcium chloride (1 to 2%) and ammonium chloride (0.5 to 2%) have been traditionally added as percentages of rations to increase water intake and produce acidic urine, with inconsistent results.

The traditional addition of these salts as a simple percentage of the diet without consideration for the components of the total ration may lead to inconsistent and unsuccessful maintenance of low urinary pH. The concept of DCAD states that with increased cations in the diet, alkalotic tendencies will occur. Conversely, increased anions in the diet have acidifying potential. Different commercial diets are commonly formulated using various commodities, and these commodities are interchanged regularly in feed preparation based on availability. If a feedstuff of a particular batch of feed is higher in cations, or anionic salts are fed in conjunction with a highpotassium forage, the DCAD of the diet will be raised and urinary acidification may not occur, despite the addition of the standard dose of anions. This one-dose-fits-all method may be the major cause of sporadic urolith formation in animals being fed anionic salts. The use of DCAD balancing for goats and urolithiasis is mentioned as a recommendation in some sources, and it is recommended that high cation-containing feedstuffs such as alfalfa and molasses should be avoided. Few controlled studies for target DCAD levels currently exist, but a DCAD of 0 mEq/kg appears to achieve urine pH of intact and castrated goats of less than 6.5. To accurately assess the efficacy of salts in the diet, whether DCAD balanced or not, owners should be encouraged to periodically assess urine pH at home.

Early castration is commonly thought to reduce the positive influence of testosterone on urethral diameter as well as diminish normal prepucial to penile attachments that are present in the neonate. Delaying castration in pet animals may serve to increase urethral diameter as well as increase the ability to examine the penis. Prophylactic removal of the vermiform appendage in young small ruminants may also serve to reduce the likelihood of obstruction.

Grazing of females on pastures which have high silica content of soil and plants is preferred to the grazing of males on these pastures. If males are to be grazed in these locations, water intake should be encouraged by maintaining desirable and accessible water sources and supplementation of anionic salts.

Summary

For prevention of urolithiasis, major efforts should be focused on reducing the grain and increasing the for-

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age composition of the diet. A 2:1 Ca:P ratio should be attained, magnesium lowered to less than 0.6%, and anionic supplementation or DCAD balancing should be considered. Palatable, fresh water should be consistently available at temperatures which encourage consumption. Because urolithiasis is multifactorial, it is difficult to achieve consistent results from preventative strategies and can be very frustrating for producers and veterinarians. With adherence to the above goals, significant reductions in clinical cases can be achieved. Veterinarians should seek input from FARAD to determine withdrawal times for analgesics and sedatives used in an extra-label manner.

References available upon request.

Pig C-sections

In my experience, swine presenting in dystocia are often in a compromised state. Particularly in the case of pet pigs, owners often lack awareness of the normal birthing events and monitoring of livestock, resulting in delays in presentation to the veterinarian. Owners of show pigs tend to be more knowledgeable, and commercial swine are rarely presented to a veterinarian for dystocia.

The most common cause of dystocia in swine is primary uterine inertia, followed by maternal undersize. Uterine inertia can usually be resolved with oxytocin, calcium and manual assistance. That works in commercial and show pigs, but is more challenging in potbellied pigs due to their small pelvic size. If you can't get your hand in to rule out obstructive dystocia, oxytocin and calcium are absolutely contraindicated. So, we often go straight to c-section on those.

We offer every client the choice of injectable or gas anesthesia with monitoring. The costs are vastly different, but we want the clients to make an informed decision. For injectable, we use a combination of xylazine (1mg/kg) or butorphanol (0.1mg/kg) with midazolam (0.2mg/kg) and ketamine (5mg/kg). Preoperatively, we also administer ceftiofur CFA (swine product) and flunixin. We have a chart for these drugs by weight to take the calculations out of it.

We place the gilt or sow in right lateral recumbency and may do either a lumbosacral epidural or a simple line block with 2% lidocaine. The approach is paramammary, just lateral to the left mammary chain. Skin and muscle are incised with a scalpel, while my preference is to open peritoneum with an initial protected stab incision with a scalpel, followed by opening with scissors.

The uterus is located and exteriorized. Particularly if you know or are suspicious of dead piglets, I recommend placing an impervious drape around the exteriorized uterus. Palpate the piglets and incise over the pig most distal in the uterine horn. Piglets can then be extracted by "milking" them towards the incision. The second uterine horn may need an additional incision based on the arrangement of the piglets.

After extraction, we spend a good amount of time lavaging the uterus and removing blood clots. This is repeated after we suture the uterus. The uterine incision(s) are closed in a Utrecht inverting pattern using 2-0 PDS on a taper needle. If the uterus is very contaminated, I recommend a second layer closure on the uterus. The clean uterus is replaced into the abdomen.

We perform an initial closure of the body wall with 2-0-0 PDS of the peritoneum and muscle layers using a taper needle in a simple continuous pattern. For a large gilt or sow, I would recommend closing in 2 to 3 runs of suture. A second layer of the fat/subcutaneous tissue is then closed, followed by the skin in a Ford Interlocking or other suitable skin pattern.

We allow the preoperative ceftiofur to complete the postoperative antimicrobial protocol and provide meloxicam orally at 0.4mg/kg PO q 24 hours. We often find that some benefit from opioid pain control, but pigs are very susceptible to opioid-induced ileus and constipation and will go off feed. So, if we provide opioid pain management (0.1mg/kg morphine IM q4-8h), we will prophylactically use canned pumpkin, Hill's a/d and sometimes lactulose to keep things moving along. Veterinarians should seek input from FARAD to determine withdrawal times for analgesics and sedatives used in an extra-label manner.

Pregnancy Toxemia

Pregnancy toxemia in sheep and goats is one of 3 syndromes falling under the heading of hepatic lipidosis. Thus, its pathophysiology is related to that of fat cow syndrome in dairy cows and protein-energy malnutrition in beef cows. The underlying cause of each of these syndromes is a negative energy balance, typically occurring at a time of metabolic drain and/or poor feed availability. In sheep and goats, this condition most often occurs in the last 2 to 4 weeks of gestation in dams carrying multiple fetuses. Contributing to the negative energy balance in late-gestation does and ewes is the increased demand of fetal growth, decreased abdominal space, poor feed availability (winter), cold, shearing stress, lack of exercise, stress of movement, and intestinal parasitism.

Clinical Picture

Affected dams typically present anorexic, depressed, recumbent and may have significant neurologic signs. Diagnosis is most easily based on the presence of ketonuria (>60 mg/dL). Hypoglycemia is variably present and it is not recommended that a normal or high blood glucose be used to rule out pregnancy toxemia. Other laboratory findings may include metabolic acidosis, hypocalcemia, azotemia, elevated GGT and AST, hypokalemia, increased FFAs, increased beta-hydroxybutyrate [BHB] (>5-7 mmol/L), increased NEFAs (>0.4 mEq/L), and stress neutrophilia. I strongly recommend that a fecal flotation be run on all cases, as I find most are heavily parasitized, contributing to the negative energy balance.

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When I approach treatment of an individual case of pregnancy toxemia, my first question to the owner is, "Who is the bigger priority to you: the doe/ewe or the kids/lambs?" If the dam is the priority, induction of parturition should be strongly considered. Goats are CL-dependent throughout pregnancy, while in sheep, the placenta takes over at about day 50 of gestation. In goats, 5 to 10 mg of dinoprost or 100ug/45 kg cloprostenol will cause parturition in 27 to 55 hours. In ewes, 20 mg of dexamethasone will result in parturition in 48 to 72 hours. In both species, I typically use both a PG and dexamethasone to expedite delivery (12 to 24 hours) and to prepare the lungs of kids for delivery. Cesarean sections may be considered, but dams should be stabilized prior to surgery. Dams with pregnancy toxemia experience a high rate of dystocia and offspring are often born weak, with high rates of failure of passive transfer. When owners want to maintain the pregnancy, treatment is determined by severity of signs and owner preference. Dams which are still eating some and are still able to stand up on their own may be maintained with oral medications. Once animals are anorexic or recumbent, however, intravenous maintenance is usually necessary.

Treatment and Monitoring

Conservative therapies include oral propylene glycol (30 to 60 mL q 12h), increase in compact, high-energy nutrition, B vitamins, 15 to 20g NaHCO3/50mL water orally, and nursing care. High-energy calf scour rehydration solutions may also be administered. For more severely affected, I generally place a catheter in the jugular vein and begin polyionic fluids with 2.5% or 5% dextrose and 10 to 15 mL B complex vitamins/L fluids. My preference is to administer this as a constant infusion, however, 100 mL boluses of 50% dextrose q 6 to 8 hours may be substituted. Other additives may include 25 to 75 mL/ L of calcium borogluconate and 10 to 30 mEq/L KCl / L. Urine glucose and ketones should be monitored to assess for need to reduce rate of dextrose administration. Rumen transfaunation (1 to 2 pints) may be considered, particularly if animals have been previously treated with propylene glycol. Insulin (PZI) at 0.4 u/kg SC q 24 h for 3-4 days or regular insulin at 0.1u/kg SC once (with glucose monitoring) increases glucose utilization and assists with reducing hyperkalemia in acidotic animals.

Monitoring throughout treatment should include serum electrolytes, acid/base balance, urine glucose and ketones and fetal viability via ultrasound. There is a good prognosis for both dam and fetuses if cases are treated early and aggressively, when dams are still ambulatory and have only a small reduction in appetite. The prognosis is poor for dams who are recumbent, have total anorexia, become ill prior to 2 weeks prior to due date, or have clinicopathologic evidence of severe hepatic lipidosis and renal failure.

Prevention

Prevention of pregnancy toxemia should first focus on obtaining an appropriate body condition score at breeding (3/5), maintaining that through parturition (3.5/5). Highenergy feeds (small grains, legumes) should not be provided in the first 4 months of pregnancy, with 8 to 10% protein hays provided. In the last month to 6 weeks of gestation, concentrates should be introduced into the diet, and increased over a 2-week period to provide increased energy in the last 2 to 4 weeks of gestation. Grazing on lush spring pasture, legume hays, and alfalfa pellets may also be used to provide this additional energy. In late gestation, owners may, for example, plan for 3.5 to 4# of good quality hay (>10% protein, >55% TDN) and 1.5# concentrate (whole shelled corn) once daily, increasing concentrate from 0.5#/head/day to 1.5#/head/ day over about 2 weeks. Severe winter stresses increase energy requirements and concentrates should be increased to 2 to 3#/head/day, divided into 2 feedings, to provide for this increased metabolic strain. Other strategies that have been used include ultrasound pregnancy examination (45 to 90 days) and separation of dams with multiple fetuses for increased feeding, monitoring for presence of urine ketones, and the determination of BHB levels in plasma. Pooled plasma may be used for herd screening of BHB, with values of 0.8 mmol/L indicating adequate energy intake, 0.8 to 1.6 mmol/L indicating inadequate dietary energy and values >1.6mmol/L indicating severe malnutrition.

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