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Diseases of the Gastrointestinal System

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In the sheep or goat, the gastrointestinal system is arguably more prone to disease than any other body system or structure. There is no substitute for a thorough physical examination in trying to determine which body systems of a sick animal are affected; this is true especially with diseases of the gastrointestinal system. A complete physical examination should include palpation for body condition, assessment of abdominal shape and rumen motility, observation of the consistency of the stool, and evaluation for the presence of bloat. Rectal palpation cannot be performed in sheep and goats, however, so localization of a disease process to a particular segment of the gastrointestinal system can be difficult. Therefore ancillary diagnostic procedures may be needed to characterize gastrointestinal diseases properly.

DIAGNOSTIC PROCEDURES

Basic Laboratory Studies

Clinicopathologic data from laboratory studies consisting of a complete blood count (CBC), serum biochemical evaluation (SBE), and urinalysis can be helpful in eliciting the presence of gastrointestinal disease, developing a prognosis and plan for treatment, and monitoring response to treatment. A CBC rarely identifies a specific disease but can be helpful in evaluating the severity of dehydration, anemia, and hypoproteinemia. The clinician must take care to interpret the packed cell volume (PCV) and total protein in light of the hydration status of the animal as noted on physical examination. An anemic or dehydrated hypoproteinemic animal may have normal PCV and total protein values. Both the CBC and SBE can be helpful in determining the presence and severity of an inflammatory disease process. Changes in the total and differential white blood cell counts indicate acute or chronic inflammation; increases in globulins or fibrinogen suggest a chronic inflammatory disease. Low protein levels, especially of albumin, can point to chronic blood loss from intestinal parasitism or infiltrative bowel disease. Liver disease should be suspected if liver enzyme or bilirubin levels are elevated. Of note, however, liver enzyme concentrations can be normal in chronic liver disease. Also, albumin levels rarely drop in ruminants with liver disease as they do in other species.[1] Changes in electrolytes can occur with gastrointestinal disease, especially if affected animals are anorexic. Electrolyte measurements also are helpful in formulating a treatment plan. Hypochloremia and metabolic alkalosis occasionally occur in abomasal disease. A mild hypocalcemia may be encountered in some small ruminants with gastrointestinal atony. Many animals with gastrointestinal disease are dehydrated, azotemic, and possibly hypoproteinemic; therefore it may be helpful to rule out urinary tract disorders in these cases.

Normal ranges for clinicopathologic laboratory values are available in Appendix 11 Tables A-D and also have been published in several other textbooks.[2-5] However, clinicians would do well to learn the normal values, especially serum biochemistry values, established by the laboratory most commonly used for analysis in their practice.[5]

Rumen Fluid Analysis

Analysis of rumen fluid can help differentiate among diseases of the forestomachs. An appropriately sized orogastric tube can be passed through the oral or nasal cavity for fluid collection (Figure 5-1). For this procedure, proper restraint of the animal, using a mouth speculum to prevent chewing of the tube if it is passed orally, is essential. If the tube is chewed, its roughened surface may damage the esophagus, and parts of a broken tube can be swallowed. Rumen fluid also can be collected using percutaneous rumenocentesis.[1,6-10] For this percutaneous technique, a 16-gauge needle is inserted in the rumen through the abdominal wall caudal to the xyphoid and to the left of midline. The clinician then aspirates fluid with a syringe. Local anesthesia and sedation of the animal may be necessary. This technique avoids the saliva contamination that can occur during collection with an orogastric tube, and it appears to be less stressful. Rumenocentesis carries a slight risk of peritonitis, but this risk can be minimized with immobilization of the animal through proper restraint.

Figure 5-1 Passage of an orogastric tube through a mouth speculum made from a polyvinylchloride (PVC) pipe. To avoid oral and esophageal trauma, the animal should be well restrained, and the tube should be lubricated and passed slowly down the esophagus.

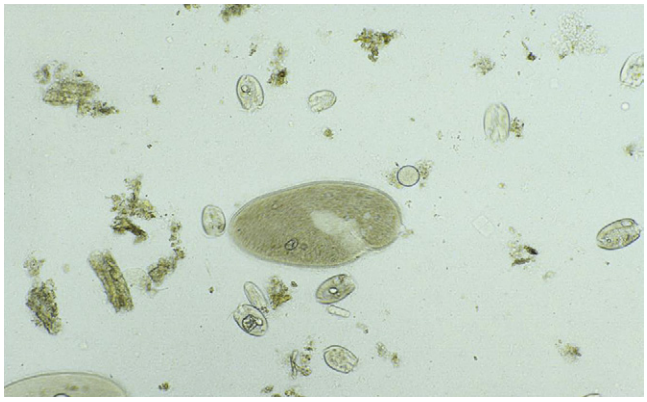

Figure 5-2 Fluid obtained by rumenocentesis should be examined for both bacteria and protozoa. A drop of rumen fluid is placed on a microscopic slide and viewed under a coverslip. At low power (40×), normal rumen fluid will be observed to contain 35 to 40 organisms/field from several populations of protozoa, as seen here. Both low numbers and loss of motility signal a need for medical intervention or transfaunation.

Percutaneous rumenocentesis should not be performed on pregnant females.

After the fluid is collected, it can be analyzed for color, odor, pH, protozoal species and motility, methylene blue reduction time (MBR), Gram staining characteristics, and chloride levels (Figure 5-2). Normal values are listed in Table 5-1. Anorexia may cause the fluid to appear darker, the pH to increase, and the number and motility of protozoa to decrease. A gray color, low pH, and dead or no protozoa are seen in rumen acidosis from grain overload. The MBR is prolonged with any type of indigestion/digestive disorder. Large numbers of gram-positive rods (*Lactobacillus* species) also may be seen in rumen acidosis. Elevated rumen chloride concentrations indicate an abomasal or proximal small intestinal obstruction (either functional or mechanical).

Abdominocentesis

Abdominocentesis is useful in discerning the causes of fluid distention in the abdomen. Two methods can be used. The first technique involves tapping the lowest point of the abdomen slightly to the right of midline; it is useful in ruling out a ruptured bladder as the cause of general ascites[1,11] (Figure 5-3). The clinician should take care to avoid the prepuce in males and mammary veins in females. The second technique is useful if peritonitis is suspected. Because localized peritonitis is more common than generalized peritonitis, four sites are tapped.[12] The two cranial sites are slightly caudal to the xyphoid and medial to the milk veins on both sides. The two caudal sites are slightly cranial to the mammary gland and to the left and right of midline. For either technique, manual restraint with sedation is recommended; the use of real-time ultrasonography may help locate fluid pockets.

A 20-gauge needle or teat cannula can be used for fluid collection.[11] The clinician should prepare the site using sterile technique and provide local anesthesia when a teat cannula is to be used. Fluid should be collected in a small ethylenediamine tetraacetic acid (EDTA) tube for analysis and a sterile tube for culture. Abdominal fluid can be difficult to obtain because of the small amounts normally present in both sheep and goats. It is important to minimize the ratio of EDTA to fluid in the sample, because EDTA can falsely elevate protein levels. Using EDTA tubes made for small animals or shaking excess EDTA out of large tubes resolves this problem. Normal culture results are similar to those for cattle (clear, colorless to slightly yellow, 1 to 5 g/dL protein, less than 10,000 cells)[12] (see Appendix Table H). Cytologic examination is needed to characterize the cell population and assess for the presence of phagocytized bacteria.

Radiography

Radiography of the abdomen can be performed in small ruminants using small animal techniques. In adults, the rumen normally fills the entire abdomen. Radiography can detect gas distention of the small intestine, abdominal fluid, and foreign bodies.[12,13] Contrast techniques are useful for diagnosing atresia of the rectum or colon. Unlike in other small animals, contrast techniques are not practical for characterizing small intestinal problems in sheep and goats, because the rumen dilutes and slows passage of the contrast media.[14]

TABLE 5-1 Normal Rumen Fluid Characteristics of Sheep and Goats

Characteristic	Normal Finding
Color	Green
Odor	Aromatic
pH*	6.5 to 7.5
Protozoa†	Mixed sizes and species rapidly moving
Methylene blue reduction time‡	3 to 6 minutes
Gram stain	Gram-negative rods predominate
Rumen chloride	Less than 25 to 30 mEq/L

Data from Nordlund KV, Garrett EF: Rumenocentesis: a technique for collecting rumen fluid for diagnosis of subacute rumen acidosis in dairy herds, Bovine Pract *28:109, 1994; Keefe GP, Ogilvie TH: Comparison of oro-ruminal probe and rumenocentesis for prediction of rumen pH in dairy cattle,* Proceedings of the 30th Annual American Association of Bovine Practice Convention, *1997, p 168; and Smith MC, Sherman DM:* Goat medicine, *ed 2, Ames, Iowa, 2009, Wiley-Blackwell.*

**Use pH paper with at least 0.5-unit gradations.*

†Place a drop of fluid on a warm slide and cover with a coverslip. Examine under 100× magnification.

‡Mix one part 0.03% methylene blue to 20 parts rumen fluid. Measure time for blue color to clear to match a control tube of fluid.

Ultrasonography

Ultrasonography can be used to provide better characterization of abdominal distention, internal and external abdominal masses, and gross lesions of the liver. With this imaging modality, ascites can be differentiated from fluid in the intestinal tract, and gas distention of the intestines can be differentiated from fluid distention. The normal ultrasonographic examination of the liver in sheep and goats has been described.[15,16] The liver can be viewed on the right side from the seventh or eighth rib caudally to the 13th rib (Figure 5-4, *A* and *B*).

Ultrasonography also can be used to guide tissue sampling for biopsy of other organs or masses and to locate pockets of fluid.

Laparoscopy

Laparoscopy more commonly is used as a reproductive tool, but it also can be used diagnostically as an alternative to exploratory laparotomy in small ruminants.[17-21] General anesthesia is recommended to allow a greater degree of inflation of the abdominal cavity for a more thorough examination, but laparoscopy can be done with use of sedation and local anesthesia at portal incision sites.

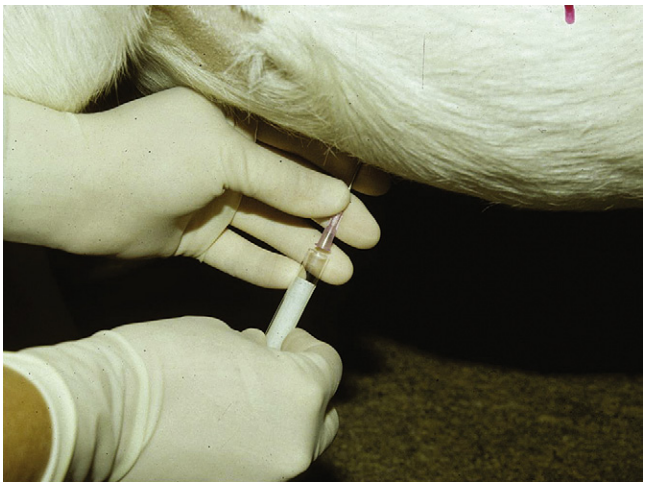

Figure 5-3 Ventral and caudal sites for performing abdominocentesis. The *needle* indicates the ventral site. The caudal site is the *clipped area* below the flank.

The technique for laparoscopic exploration of the abdomen in cattle and llamas can be modified for use in sheep and goats.[18-21] Laparoscopic evaluation of the abdominal cavity is usually done through a ventral approach, with the animal secured in dorsal recumbency. The abdominal cavity can be inflated with CO_2 delivered by a needle or teat cannula or after placement of a laparoscopic cannula. A time-saving method is to make a "bite" through the skin and into the external rectus sheath with a suture, which can be pulled tight in order to tense the body wall. A stab incision can then be made in the skin and external rectus sheath before introduction of a guarded trocar into the abdominal cavity while tension is applied to the abdominal wall using the previously placed suture. Next, the laparoscope is placed through the trocar and the abdomen inflated under visualization through the scope. The cannula is then placed in the inguinal area as described for laparoscopic insemination (Chapter 8). This technique allows a more efficient use of time and minimizes the likelihood that the omentum will be "ballooned."

Laparoscopic placement into the right side allows visualization of most of the abdominal organs (Figure 5-5, *A* to *C*). Obviously, the clinician should avoid the rumen when introducing the laparoscope into the abdomen. This procedure may be enhanced by lowering the head or rear of the animal, allowing better visualization of the entire abdomen. Visualization of the abdominal cavity and the ability to manipulate organs will be greatly improved by fasting the animal for 24 to 48 hours or at least decreasing the bulk in the diet. Respiration must be monitored closely, and assisted ventilation should be available during this procedure, because inflation of the abdomen and lowering of the head can put pressure on the diaphragm.

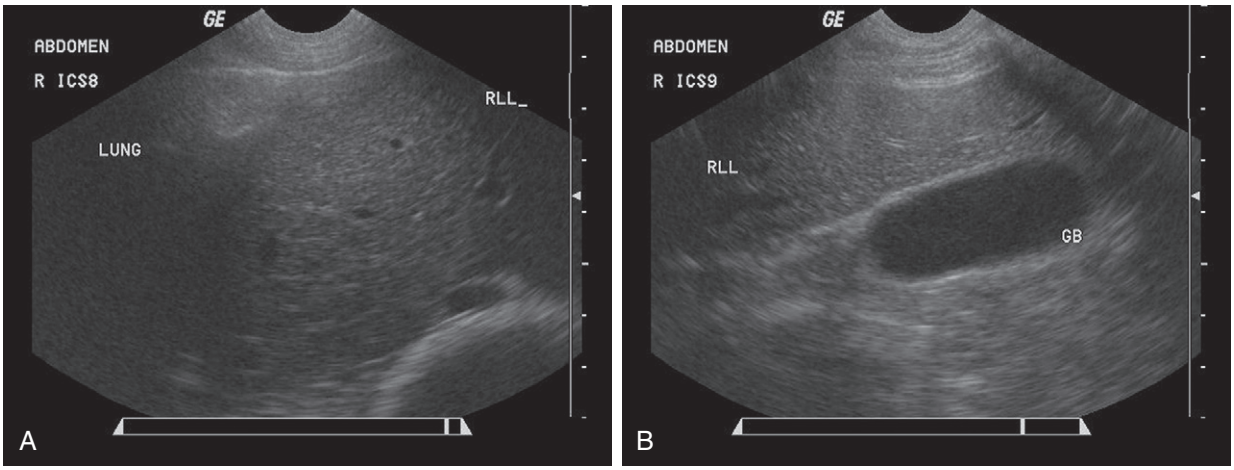

Figure 5-4 **A,** Ultrasound image of the right abdomen obtained from the right eighth intercostal space in a 3-year-old LaMancha cross doe, showing the right liver lobe with the characteristic hepatic and portal veins, represented by the small, tubular anechoic structures within the liver parenchyma. The ventral border of the lung is seen on the *left* side of the image. This ultrasound scan was obtained using a 7-MHz microconvex transducer. Dorsal is to the *left* of the image. **B,** Ultrasound image of the right abdomen obtained from the right ninth intercostal space of the same animal as in **A,** demonstrating normal right liver lobe and gallbladder. The gallbladder appears as an anechoic, fluid filled structure directly adjacent to the right liver lobe. This ultrasound scan was obtained using a 7-MHz microconvex transducer. Dorsal is to the *left* of the image. *(Courtesy Dr. Karine Pader, Purdue University.)*

Exploratory Laparotomy

Exploratory laparotomy can be a valuable diagnostic tool in evaluating gastrointestinal diseases when other tests indicate abdominal disease. In some cases, therapeutic surgical procedures can be performed at the same time. The technique of exploratory laparotomy used in cattle can be adopted for sheep and goats with the understanding that these animals are more likely to lie down during surgery; therefore standing surgery should be attempted only rarely.[22]

For this procedure, small ruminants should be heavily sedated or placed under general anesthesia. Animals that show signs of postoperative pain, anorexia, and depression should be treated accordingly with a nonsteroidal antiinflammatory drug (NSAID) (e.g., flunixin meglumine, 1.1 to 2.2 mg/kg intravenously [IV]).[12] The decision to use perioperative and postoperative antimicrobial agents should be based on the conditions under which the surgery is performed and the diagnosis made at surgery. Antimicrobial agents are not necessary for elective exploratory surgery performed aseptically, in a hospital setting, and without complications. However, they are indicated in field conditions, if infection is already present, and if the intestinal tract is opened. A combination of ceftiofur (1.1 to 2.2 mg/kg intramuscularly [IM] or subcutaneously [SC] twice a day) and procaine penicillin G (22,000 international units [IU]/kg IM twice daily) can be administered until culture results indicate an absence of microbes (see also Appendix 1).

Liver Biopsy

Liver biopsy in sheep and goats is performed using the same technique and instruments as in cattle and llamas.[23,24] Sedation and ultrasound guidance are recommended.[24] The recommended biopsy site is in the ninth to tenth intercostal space slightly above an imaginary line from the tuber coxae to the point of the elbow (Figure 5-6).

The site should be surgically prepared, and a local anesthetic (2% lidocaine hydrochloride) infused subcutaneously. A small scalpel blade is used to make a stab incision through the skin. A 14-gauge, 11.5-cm liver biopsy instrument is inserted through the incision and the intercostal muscles and into the liver. The biopsy instrument should be directed toward the opposite elbow in most cases, but the use of real-time ultrasonography can help determine the direction and depth needed (2 to 4 cm). The clinician should attempt to avoid large vessels along the caudal border of the ribs. On reaching the liver, the clinician will note a slight increase in resistance. Samples can be submitted for culture (in a sterile plastic or glass vial or tube), histopathologic study (in formalin at a 10:1 ratio of formalin to tissue), or mineral analysis (in a plastic tube). When performing a liver biopsy for mineral analysis, the clinician should rinse the biopsy site with distilled and deionized water after sterile preparation to minimize sample contamination. Samples for mineral analysis should not be placed in formalin.

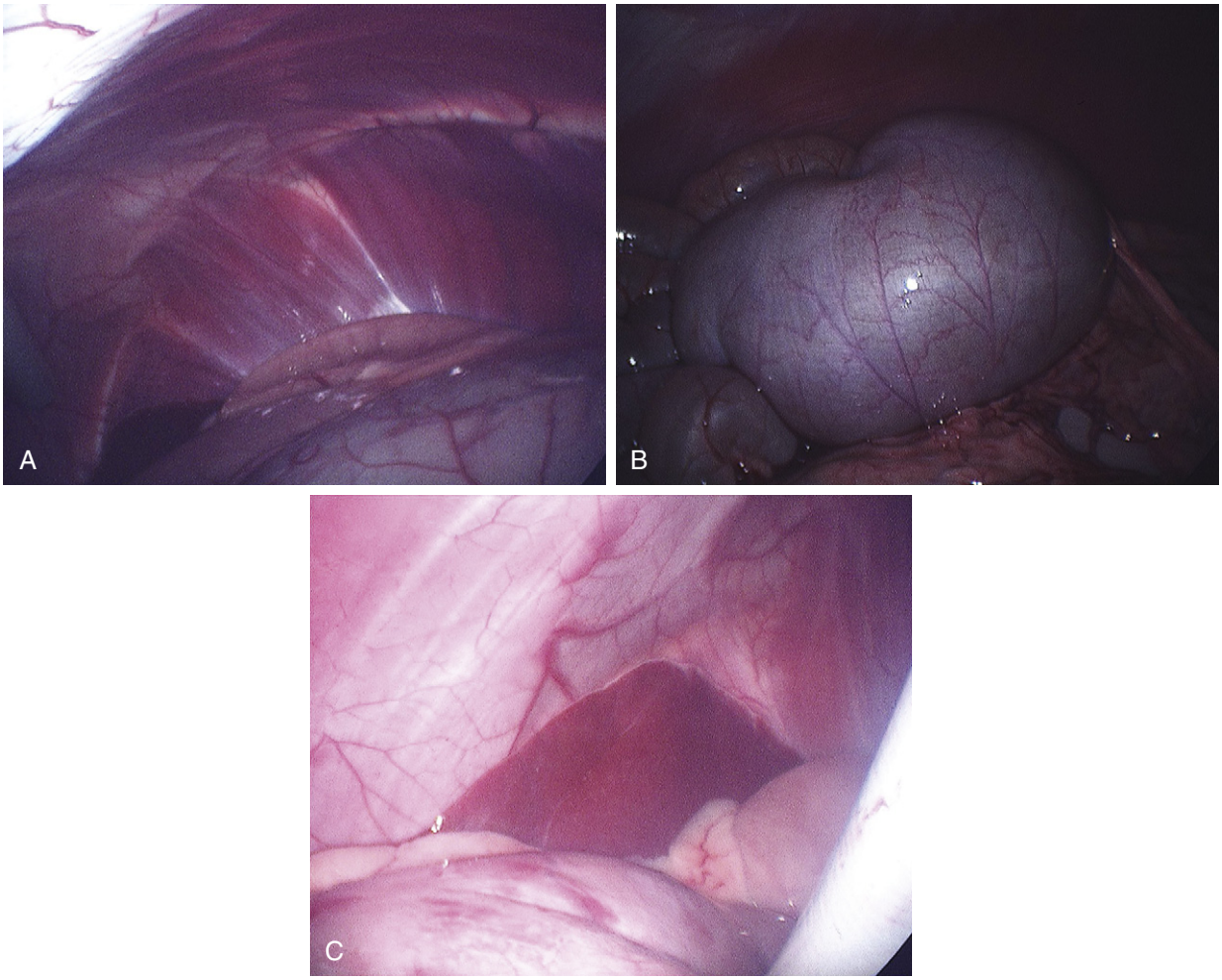

Figure 5-5 A laparoscopic examination (peformed using a 10-mm-diameter direct vision scope) of the abdomen in a 2-year-old Pygmy buck. **A,** The muscle fibers of the diaphragm are evident cranially in the *center* of this photograph. A small part of the liver is in the *lower left* of the image. **B,** The larger organ in the *center* of this photograph is the cecum. It normally appears darker in comparison with other portions of the intestine and contains ingesta of a doughy consistency. **C,** This photograph shows part of the liver on the right body wall.

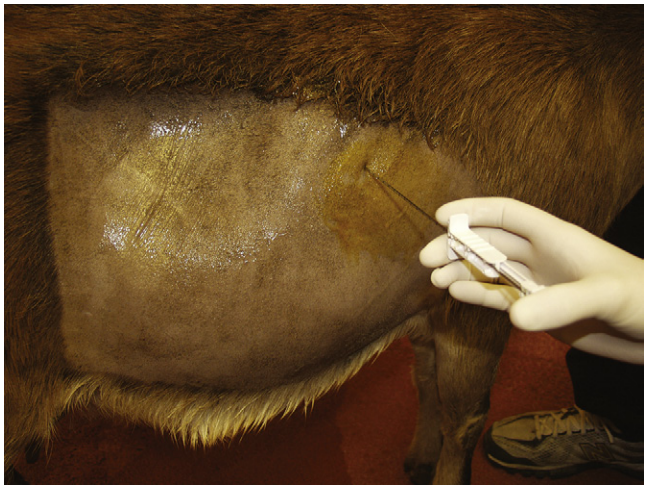

Figure 5-6 Liver biopsy: After the skin is clipped, anesthetized, and aseptically prepared, the surgeon makes a stab incision in the skin and introduces a 14-gauge biopsy needle.

Closure of the skin incision can be accomplished by suturing or stapling, or if it is small enough, the wound can be left alone to heal by second intention. The clinician or the keeper should apply fly repellent to the area. The animal's production record should show *Clostridium* prophylaxis; if it does not, vaccination during or before the biopsy is indicated.

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DISEASES OF THE FORESTOMACHS

Bloat

Bloat is less common in small ruminants than in cattle, with goats being affected less commonly than sheep. *Bloat* is the accumulation of either free gas or froth in the rumen, which causes rumen distention. The causes of bloat can be divided into three categories[1,2]:

Frothy bloat—caused by diets that promote the formation of stable froth

Free gas bloat—caused by diets that promote excessive free gas production

Free gas bloat—caused by failure to eructate

Pathogenesis

Frothy bloat usually is associated with the ingestion of legume forages or hay (particularly alfalfa) and with grazing on lush cereal grain pastures, but it also may occur with high-grain diets.[3] In the case of frothy bloat from a fine-ground diet (usually corn), mucoprotein released from rumen protozoa stabilizes the foam at a low pH. In legume-associated frothy bloat, plant chloroplasts released into the rumen trap gas bubbles. Regardless of the form of frothy bloat, the small bubbles fill much of the rumen, preventing clearance of the rumen's cardia and resulting in a cessation of eructation. Free gas bloat also occurs with grain diets, especially if the animals are not adapted to the diet. Failure to eructate has a variety of causes. Physical obstructions of the esophagus such as with choke or swollen mediastinal lymph nodes can cause free gas bloat. Any disease of the rumen wall may result in impairment of contractions and eructation. Hypocalcemia, endotoxemia, pain, peritonitis, and some pharmaceutical agents (especially xylazine) all produce conditions that interfere with rumen function and eructation.[1,2,4,5]

Clinical Signs

Clinical signs of frothy bloat and free gas bloat from either food intake or physical obstruction of the esophagus usually are more severe and immediately life-threatening than those associated with bloat due to rumen wall diseases and systemic influences. Abdominal enlargement occurs, particularly in the dorsal left paralumbar fossa. This ruminal enlargement may be subtle in sheep or Angora goats with full fleece. Signs of colic and anxiety are common. The rumen may be either hypomotile or hypermotile. Respiratory distress is obvious, with mouth breathing evident in some animals; death can ensue if the bloat is not treated.[3]

Diagnosis and Treatment

Presence of bloat constitutes a medical emergency, so diagnosis and treatment should occur almost simultaneously. If the animal is not in immediate danger of dying, an orogastric tube can be passed. Most cases of free gas bloat are relieved with passage of the tube. A thorough history and complete physical examination are then indicated to find the cause of the bloat. If the bloat is not relieved with passage of an orogastric tube, the tube should be removed and examined for evidence of froth. Frothy bloat can be treated with poloxalene (44 mg/kg) or dioctyl sodium sulfosuccinate (DSS) (28 mL [1 oz]) delivered by orogastric tube. The froth encountered in frothy bloat, caused by the ingestion of finely ground grain, has a pH of less than 5.5. If frothy

bloat develops while animals are being fed concentrates, mineral oil (100 mL) may work better. Peanut oil (20 to 50 mg/kg), vegetable oil (100 to 200 mL), and hand soap (10 mL) also have been recommended in emergency situations.[3]

If the animal is in severe respiratory distress, the clinician should insert a trocar or large needle into the rumen at the paralumbar fossa. If gas does not escape, or froth is seen coming out of the trocar, an emergency rumenotomy is indicated (see later under "Rumenotomy").[3] With occurrence of bloat in multiple animals of a pastured group, the entire group should be removed from the pasture and reintroduced slowly after gradual acclimation. If only one or two cases of bloat are encountered, the healthy animals can remain on the offending pasture, but grazing should be limited, to ensure gradual acclimation.

Prevention

Prevention of frothy bloat involves limiting access to offending pastures or feedstuffs; providing supplemental feed and providing poloxalene in mineral supplements; and adding ionophores to the ration or supplement. When grazing or consuming legumes as green chop animals should be introduced to the feed or pasture slowly, preferably over 2 to 3 weeks. Animals should be closely monitored after a frost and during the rapid growth phase of plants, because legumes, particularly alfalfa, may be more likely to cause bloat at this time. Certain varieties of legumes that are designed for intensive grazing systems (e.g., Alfagraze) should be planted and managed in a manner that decreases the incidence of bloat (e.g., limited or creep grazing). Feeding dry, stemmy hay for 1 to 2 hours before allowing access to the legume pasture also may help minimize occurrence of bloat. Grass-legume pastures in which legumes are limited to less than 50% of the forage are safer but can still pose a problem with animals that are selective grazers. Grazing legumes with high leaf tannin concentrations (e.g., arrowleaf clover, kudzu) usually is safer, because tannins help break down rumen foam. The inclusion of poloxalene (10 to 20 mg/kg daily) in the feed or mineral supplement is useful in preventing frothy bloat. If poloxalene supplements are used, keepers should feed them for 1 to 2 weeks before moving animals onto a problem pasture.

Free gas bloat from concentrate feeds can be controlled by slow introduction to these feeds to allow for rumen adaptation and by the inclusion of ionophores in the diet.[1] Monensin (15 mg/head/day in ewes and 1 mg/kg/day in goats) and lasalocid (0.5 to 1 mg/kg/day in sheep and goats) both decrease the formation of free ruminal gas. By enhancing propionic acid formation, these drugs not only reduce the amount of methane produced in the rumen but also improve the efficiency of nutrient assimilation from feedstuffs.

Bloat in lambs and kids can have the same causes as in adults but also can be caused by improper milk feeding. Overfeeding, feeding of large infrequent meals, and feeding spoiled or cold milk all have been associated with bloat in lambs and kids.[6] Rapid overdistention of the abomasum and improper chemical or physical composition of milk replacers both will inhibit rumen motility, leading to bloat. Even though the feeding of cold milk has been associated with bloat, the practice can be used effectively in orphan feeding programs. Lambs and kids tend to limit their intake of cold milk after they have become accustomed to a free-choice feeding system that delivers refrigerated milk. Milk usually is placed in the rumen when animals are tube-fed; this may result in milk spoilage.[1,6]

Simple Indigestion

Simple indigestion is a mild form of upset of reticulorumen function caused by a change in feeding routine. Such changes typically involve alterations in the type of feed or in the amount of feed offered. The most common causes of simple indigestion are the addition of grain to the diet, an increase in the amount of grain fed, and an increase in the energy density of the diet. Examples of such dietary changes are replacing oats with corn and changing from whole to ground corn. If the changes are drastic, rumen acidosis (discussed next) can occur. Other common causes are changes in hay or pasture, consumption of moldy hay, and ingestion of weeds and toxic plants after overgrazing or drought. Clinical manifestations include mild anorexia that lasts for 1 to 2 days; mild diarrhea and bloat also may be present. Rumen fluid pH may be unchanged, increased, or decreased, depending on the inciting cause. Most animals improve with no treatment.[1]

Rumen Acidosis

Pathogenesis

Rumen acidosis is caused by the rapid rumen fermentation of highly digestible carbohydrates that are ingested in excessive amounts. Although corn commonly is implicated, other cereal grains (oats, wheat, barley) may be the offending feedstuffs, particularly if they are finely ground. The smaller the particle size, the more quickly rumen bacteria are able to ferment the carbohydrates contained in the feed. The common name for this condition is "grain overload," but breads, candy, apples and other fruits, beets, and potatoes also have been implicated as sources of the excess carbohydrates.

Rumen acidosis usually occurs in animals that have been fed predominantly forage-based rations and suddenly are given access to large amounts of highly fermentable concentrates or concentrated forms of energy. It also can occur in animals that have been receiving

concentrates previously if the amount is suddenly and drastically increased; if access is denied for a time and then suddenly returned (e.g., during weather changes or with alterations in water availability); or if ration mixing errors occur (e.g., leaving out monensin and rumen buffers).

As highly digestible carbohydrates are fermented, rumen pH drops. *Lactobacillus* species, which are lactic acid producers, proliferate in the acidic rumen environment and further lower rumen pH. As the rumen pH drops, rumen protozoa and many of the lactate users begin to die. Lactic acid production causes the osmotic pressure in the rumen to increase. Fluid is drawn from the systemic circulation into the rumen, resulting in dehydration and possibly hypovolemic shock. Lactate concentrations increase in the blood, potentially leading to systemic lactic acidosis. The lactic acid in the rumen also is toxic to the rumen epithelium. Damage to the epithelium can result in leakage of bacteria and toxins into the portal and systemic circulation. Chronic sequelae to rumen acidosis include fungal rumenitis and occasionally formation of liver abscesses.[1,7] Liver abscesses are less commonly encountered in sheep and goats than in cattle. Laminitis also can occur but may be more of a problem in sheep than in goats.[8] The severity of the disease depends on the composition of the feed, particle size, amount of feed consumed, and the period of adaptation to the diet.

Clinical Signs

Clinical manifestations vary with the amount and type of feed ingested and the time since ingestion. Signs first appear 12 to 36 hours after ingestion of the offending feed; they range from anorexia, depression, and weakness to recumbency in an animal suffering from severe circulatory shock. Dehydration usually is severe, and evidence of toxemia is present (e.g., injected mucous membranes, increased scleral injection). Colic, bilateral ventral abdominal distention, rumen stasis, and a "splashy" feel to the rumen also may be noted. Diarrhea can develop, adding to dehydration.[1,8,9] The diarrheal output can range from paste-like feces to very watery droppings with foam, occasionally with pieces of easily recognizable grain. Dehydration, lactic acidosis, and toxemia may result in ataxia, head pressing, opisthotonos, seizures, and other neurologic abnormalities.[10] The body temperature initially is elevated but may drop as the condition worsens or the animal becomes toxic. Secondary thiamine deficiency also can contribute to neurologic changes.[11]

Diagnosis

The rumen fluid pH may fall below 5.5. The fluid itself is milky gray, and particles of the inciting feed may be noted. Protozoa usually are reduced in number or absent, and large gram-positive rods (*Lactobacillus* species) may be seen on Gram staining.[9] Clinicopathologic laboratory data are consistent with dehydration (increased PCV and total protein, prerenal azotemia) and metabolic acidosis.[9] Liver enzymes (gamma-glutamyl transpeptidase [GGT], aspartate aminotransferase [AST], lactate dehydrogenase [LDH]) may be elevated on serum biochemical analysis.[1,11] The leukogram can vary in appearance, ranging from normal to a degenerative left shift, depending on the severity of the case. Urinalysis reveals an increased specific gravity.

Treatment

Treatment is aimed at correcting cardiovascular shock, dehydration, acidosis, and toxemia and removing or neutralizing the offending feedstuffs. Intravenous fluids containing 5% sodium bicarbonate should be administered.[1,12] Oral fluids are contraindicated because absorption is diminished, potentially increasing the rumen distention and worsening the animal's discomfort. NSAIDs are indicated to control the pain and inflammation of toxemia (flunixin meglumine, 1.1 to 2.2 mg/kg IV).[1,12] Oral administration of magnesium hydroxide and magnesium oxide (1 g/kg) may neutralize the acidic pH and is sufficient in mild cases. However, if much of the feed is still in the rumen, these two alkalizing agents will only work temporarily. Oral antibiotics have been recommended to kill rumen microflora and stop fermentation. We believe that these agents are contraindicated, however, because the gram-negative anaerobes that need to flourish to reestablish normal rumen microflora are susceptible to most antimicrobials effective against *Lactobacillus* species. Removing the substrate for growth of *Lactobacillus* organisms is more effective. Because orogastric tubes with large-enough bores for reflux of feedstuffs are too large for sheep and goats, rumenotomy is indicated in severe cases to remove the feed (see earlier under "Rumenotomy"). After the rumen pH is corrected, transfaunation of the rumen microflora with approximately 1 qt of rumen fluid from another small ruminant is beneficial (Box 5-1). Thiamine supplementation (vitamin B_1, 5 mg/lb SC, three to four times a day) is indicated until rumen function returns.[11] In certain instances, calcium may be indicated and can be added to the intravenous fluids (as calcium gluconate). The clinician should avoid mixing calcium salts and sodium bicarbonate.

Bacterial leakage into the rumen wall, liver, and systemic circulation makes antimicrobial therapy necessary. The systemic antimicrobial agent of choice is penicillin (procaine penicillin G, 22,000 IU/kg IM twice daily), because anaerobes are the most likely offending organisms. With aggressive treatment, the prognosis for short-term survival is good. Feed (grass hay only) and water should be limited until rumen contractions return, to prevent overdistention of the rumen. The chronic sequelae discussed previously influence long-term survival.

BOX 5-1 Collection, Handling, and Storage of Rumen Fluid for Transfaunation

COLLECTION

Collection is easiest from the rumen of a fistulated adult cow. If a fistulated cow is unavailable, fluid can be collected through a weighted orogastric tube. Alternatively, fluid can be collected from any normal ruminant at slaughter.

HANDLING

Rumen contents collected from a fistulated cow or at slaughter can be strained through gauze or cheesecloth to separate the fluid from the fibrous contents. Fluid collected through a weighted tube should be ready for storage.

STORAGE

Rumen fluid ideally should be administered immediately. However, it can be stored for 24 to 48 hours, as follows: The surface of the fluid is covered with a layer of mineral oil to maintain an anaerobic environment and the open container is refrigerated. CAUTION: Do not store rumen fluid in a closed container, because it may explode.

Prevention

Prevention involves introducing concentrate feeds slowly to allow rumen microflora adaptation. Dietary change from a lower to a higher fermentable energy concentration should occur slowly, preferably over a 2- to 3-week period. In the case of animals being fed high-grain rations (e.g., club lambs, feedlot lambs, dairy goats), buffering agents can be added to the diet. Rumen buffers may improve milk production, increase feed intake, and increase rate of gain. The crude fiber content should constitute a minimum of 20% of the diet's total digestible nutrients (TDN). For example, if the TDN is 75%, the minimum acceptable crude fiber is 15%. Crude fiber levels lower than this can be fed for short periods if the rumen is properly adapted, but problems may nevertheless occur. Sodium bicarbonate probably is the most commonly used buffer; it can be offered on a free-choice basis or included in the diet as 1% of dry matter intake. Calcium carbonate or limestone (both of which have low rumen solubility) and magnesium oxide (which has poor palatability) also can be included in the feed. Magnesium oxide should be limited to 0.5% to 0.8% of the dry matter intake.

Reticulitis, Rumenitis, and Parakeratosis

Pathogenesis

Reticulitis and rumenitis can result from chemical or mechanical damage to the mucosal lining of the reticulorumen. The most common cause of chemical damage is rumen acidosis. However, ingestion of caustic toxins also can damage the mucosa. Mechanical damage can occur from ingested foreign bodies or rumen bezoars. In cattle, viruses such as the agents of bovine virus diarrhea and infectious bovine rhinotracheitis can infect the rumen wall. Similar viruses have yet to be identified in sheep and goats.

After the mucosa has been damaged, secondary infection by bacteria or fungi can occur.[13] Previous treatment with oral antibiotics may predispose animals to development of fungal infections of the rumen wall, especially if the mucosa is already damaged. Actinobacillosis, actinomycosis, and tuberculosis rarely affect the rumen wall. Tumors of the rumen wall also have been reported.[1,14] Not all of these causes of reticulitis and rumenitis have been reported in sheep and goats, but all are potential problems.

Clinical Signs

The clinical manifestations of these diseases are vague. Anorexia and forestomach hypomotility may be the only clinical signs.

Diagnosis

Confirming a diagnosis of these diseases also may prove difficult. Samples of rumen fluid may show only changes associated with anorexia (alkaline pH, decreased numbers and motility of protozoa, prolonged MBR time; see Table 5-1 for normal values). Occasionally, fungal organisms may be seen on Romanowski (Diff-Quick)-stained slides of rumen fluid. In such cases, a diagnosis of fungal rumenitis should be made. An exploratory laparotomy and rumenotomy may be required to identify foreign bodies or masses. Rumen parakeratosis is characterized by dark, thickened, and clumped rumen papillae. It is seen mainly in feedlot lambs that consume finely ground or pelleted rations.[15] The parakeratotic rumen papillae are fragile and vulnerable to damage, which can increase the risk for development of rumenitis.[1]

Treatment and Prevention

Treatment depends on the inciting cause. Dietary changes should be made to decrease energy density and increase fiber intake. Mild rumenitis may subside with time and supportive care (i.e., transfaunation, fluid support, high-quality feed). Fungal rumenitis can be treated with oral thiabendazole, 25-44 mg/kg, when available.[16] Severe changes may lead to scarring and permanent impairment of rumen function.

DISEASES OF THE RETICULORUMEN

Traumatic Reticuloperitonitis

Traumatic reticuloperitonitis is not as common in small ruminants as in cattle, but it has been reported. Goats are affected more commonly than sheep. The overall lower incidence probably is related to the dietary habits of small ruminants, which tend to be selective grazers and do not "vacuum" the ground as cattle do. Offending foreign bodies that cause traumatic reticuloperitonitis include pieces of wire and needles.[17-19] The clinical signs are identical to those in cattle and may include anorexia, depression, colic, signs of heart failure, and evidence of draining tracts from the chest cavity. Treatment usually is difficult.

Rumen Impaction

Rumen impaction can occur after dehydration, blockage of the omasal orifice by a foreign body, sand ingestion, or consumption of diets high in fiber and low in digestibility.[20] Rumen impaction with plastic trash bags present in the environment has become a growing problem worldwide.[18] Clinical manifestations are nonspecific, but the firm rumen usually can be palpated in the left flank. The feces may be scant and dry. Oral fluids containing magnesium sulfate (60 g) may loosen impactions, but a rumenotomy is required in severe cases.[20]

Rumenotomy

To reduce rumen fill in sheep or goats requiring rumenotomy, ideally feed should be withheld for 24 hours before surgery. Such preparation usually is impossible, however, because in most cases rumenotomy is an emergency procedure. The perioperative administration of antimicrobial agents is essential, because even with meticulous technique, some contamination of the incision site and possibly the peritoneal cavity is inevitable. Because the rumen microflora is composed predominantly of anaerobic bacteria, penicillin (22,000 IU/kg) is the antimicrobial agent of choice and should be administered 2 to 4 hours before surgery. If the rumenotomy is being performed on an emergency basis, penicillin salts (potassium or sodium), which can be given by the intravenous route, provide therapeutic concentrations more rapidly than can procaine penicillin. NSAIDs (e.g., flunixin meglumine, 1.1 to 2.2 mg/kg IV) also are recommended before surgery. If necessary, treatment of cardiovascular shock and dehydration with intravenous fluids also should begin before surgery and continue until the animal is rehydrated and in stable condition (see Chapter 3).

Rumenotomy more commonly is done with the small ruminant in right lateral recumbency, because the standing patient is likely to become recumbent anyway during the procedure; either the animal will need to be restrained on some elevated surface, or the practitioner will have to operate while kneeling. The procedure can be safely done with use of local anesthesia, usually assisted by sedation in most animals. The use of general anesthesia may be considered in fractious or very valuable animals, to decrease the potential for abdominal contamination (see Chapter 18). The clinician should clip and surgically prepare a square area from 5 cm in front of the last rib to the tuber coxae, and from the dorsal midline to the lower abdomen, encompassing the entire left paralumbar fossa.

The routine skin and body wall incision is made in the middle of the left paralumbar fossa. The surgeon makes a skin incision approximately 5 cm longer than a handwidth, 5 cm caudal and parallel to the last rib. The incision is continued through the muscle layers into the abdomen. Because the abdominal wall is relatively thin, the surgeon should take care not to enter the rumen or bowel. The body wall incision must be more than adequate in size to facilitate entry of the practitioner's hand and possibly forearm, to allow exploration of the rumen and evacuation of its contents. The rumen incision will be smaller than the body wall incision after the rumen is secured to the skin. After the body wall incision has been made, a thorough exploration of the abdominal cavity should be performed before the rumen is secured to the skin. (NOTE: Exploration of the abdominal cavity is absolutely contraindicated after the rumen has been opened.) After abdominal exploration, the rumen is secured to the skin by creating a watertight seal with continuous suture. The watertight seal is critical in preventing abdominal contamination. The rumen is secured to the skin with use of monofilament (or coated) suture with minimal drag in the tissue on a cutting needle in a Cushing pattern. The first "bite" is taken in the skin at either 3 or 9 o'clock (Figure 5-7); then the second bite is taken in the rumen at that location in the opposite direction. The suture is then tied and the tail left long. To prevent or minimize leakage, the rumen suture bites should be through the seromuscular layer but not penetrate the mucosa, which could lead to leakage at closure. The Cushing pattern is continued from the midpoint of the incision to the dorsal- and ventralmost aspects of the body wall incision, where a modification resembling a W stroke is done to ensure a seal at those areas. If the practitioner starts at the 9 o'clock point and sutures dorsally, the following technique is used at the dorsalmost part of the body wall incision: The standard bite (approximately ¼ inch) is made in the rumen near the end of the body wall incision; then the suture is passed superficial and dorsal to the end of the incision by approximately 2 inches before a bite is taken in the skin toward the incision. The suture then is passed superficially to

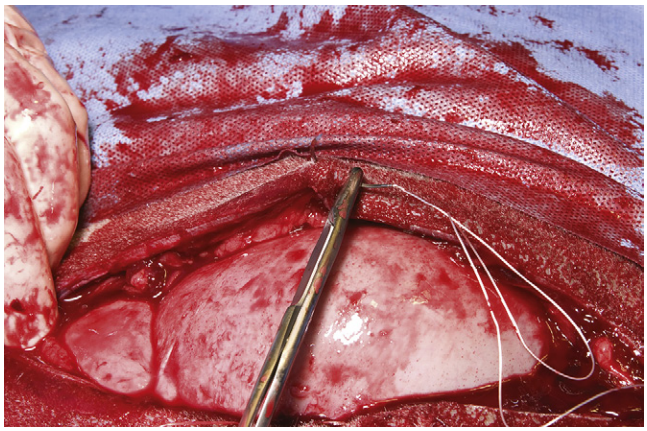

Figure 5-7 Rumenotomy: First suture "bite" in the skin at the 9 o'clock position parallel to the incision placed in a dorsal to ventral direction.

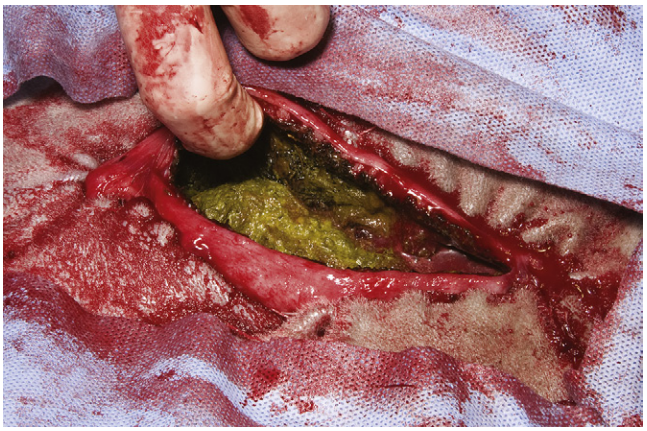

Figure 5-9 Rumenotomy: Rumen contents are visible through the rumenotomy incision.

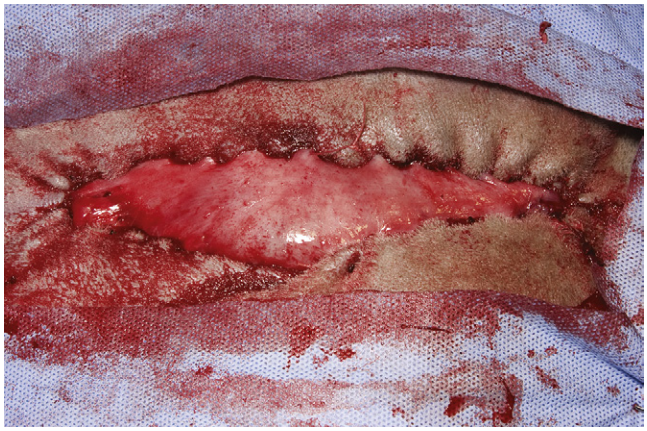

Figure 5-8 Rumenotomy: The rumen is secured to the skin with a watertight seal, ready for the rumenotomy incision.

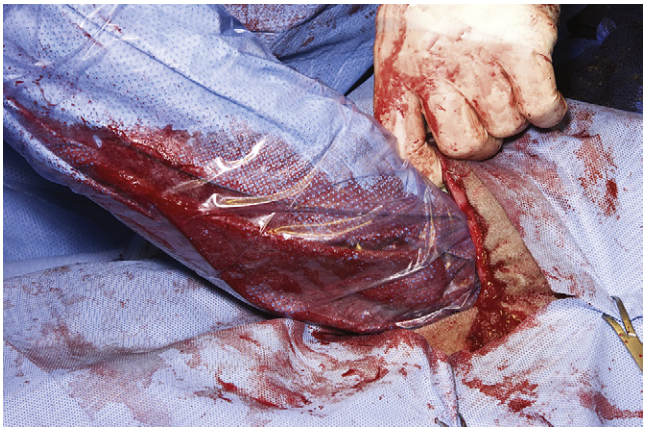

Figure 5-10 Rumenotomy: A gloved hand is used to explore the rumen and to evacuate its contents.

the dorsalmost part of the rumen, where a transverse bite (perpendicular to the suture line) is taken in the rumen. The suture is again passed superficial and dorsal to the body wall incision, where a skin bite is made parallel to and at the same level as the previous skin suture. Then the Cushing pattern is continued from the dorsalmost (12 o'clock) position down to the 3 o'clock location, where the suture is tied. The same procedure then is followed from 3 o'clock ventrally to 6 o'clock, where the dorsalmost suture pattern is repeated and continued to the originating 9 o'clock position, where the suture is tied. Two separate suture lines are used to limit the circumferential decrease in lumen size created by one suture line pulled tightly. If an assistant is available, each operator can work simultaneously on the two separate suture lines. The rumen can now be rolled over the skin to create a watertight seal (Figure 5-8). The rumenotomy incision is then made in the center of the exposed, secured rumen (Figure 5-9). Once the rumen has been secured and opened, no other modifications should be made to the rumenotomy. The rumen contents can be evacuated by hand, which will lead to significant contamination of the field—thereby showing that a watertight seal is imperative (Figure 5-10). If the contents are very liquid, a large tube can be used to siphon the rumen. In such instances, care must be taken to guard the end of the tube, to prevent occlusion of flow by suction of the rumen wall over the end of the tube.

Closure of the rumen is performed in two layers. Absorbable suture in a simple continuous pattern is used to close the rumen lumen for the first layer, and any drapes should be removed. The surgical field should be reprepared; all soiled materials (e.g., gloves, gown, drapes) are removed and replaced, and sterile instruments are readied for the second part of the closure. Absorbable suture in an inverting pattern (e.g., Lembert, Cushing) is used for the second

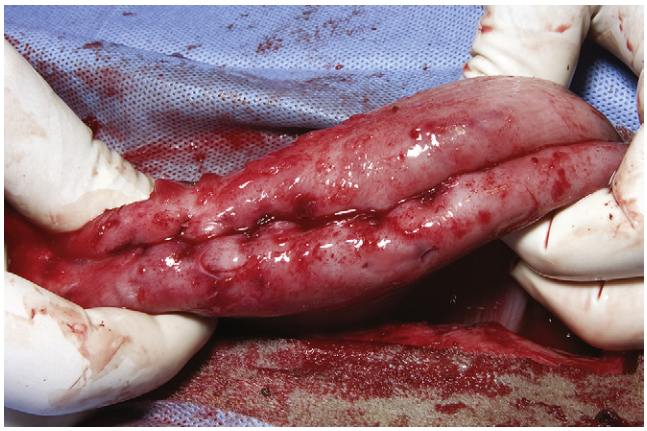

Figure 5-11 Rumenotomy: The final inverted closure of the rumenotomy incision.

layer of the rumen closure. Suturing of this second layer should start at one end of the rumen incision, and retention sutures securing the rumen to the skin are removed as needed to free enough rumen for closure. When the second layer closure is complete (Figure 5-11), the rumen is cleaned with moist sponges before being returned to the abdominal cavity. (NOTE: Exploration of the abdominal cavity at this time is associated with an increased incidence of septic peritonitis.) The muscular body wall and skin are closed in routine fashion using the practitioner's technique of choice.

The sheep or goat should be observed closely by the clinician for signs of complications, including peritonitis, incisional dehiscence, incisional hematoma, abscess, and hernia formation. Penicillin therapy (with procaine penicillin G, 22,000 IU/kg twice daily) should continue for at least 5 days. The skin sutures can be removed 10 to 14 days after surgery.

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DISEASES OF THE ABOMASUM

Abomasitis and Abomasal Ulcers

Abomasitis and abomasal ulcers in adult sheep and goats are associated with rumen acidosis or chronic rumenitis but also can be caused by infections.[1-5] Finely ground feeds, pelleted rations, systemic stress, and feeding lush forages all have been implicated. Anecdotal associations with mineral deficiency (i.e., copper) have gone unproved.

Clinical Signs and Diagnosis

Abomasitis and abomasal ulcers can be asymptomatic or manifest with a variety of clinical signs including anorexia, bloat, colic, and diarrhea. No definitive antemortem diagnostic tests are available. Fecal occult blood often is absent in ulcerative disease. Occasionally dark stool, altered appetite (e.g., wood chewing), and bruxism are seen. Thus other causes of colic should be eliminated and diagnosis based on clinical signs or postmortem findings.

Treatment

Effective therapy can be difficult to achieve. Oral medications such as coating agents must first pass through the rumen and therefore arrive at the abomasum diluted. Intravenous (not oral) ranitidine (15 mg/kg once a day) may be beneficial.[6] Herd problems of rumen acidosis may be addressed with addition of buffers to the feed.

Abomasal Hemorrhage

A syndrome of abomasal hemorrhage, bloat, and ulceration is encountered in lambs and kids 2 to 10 weeks of age. *Sarcina*-like bacteria, *Clostridium falax, Clostridium sordelli,* and *Clostridium septicum* have been isolated in many of these cases.[7-11] *C. septicum* infections of the abomasum commonly are called *braxy.*[1] Free-choice milk replacer feeding regimens, iron deficiency, and bezoars have been implicated as predisposing factors.[11,13]

Clinical Signs

The clinical manifestations of this syndrome are severe, acute abdominal distention and colic, possible ulceration, with progression to death in all cases.[7-10]

Diagnosis and Treatment

The diagnosis of this condition is by postmortem examination. Treatment in suspected antemortem cases is unsuccessful.

Prevention

Adding formalin to milk replacers and vaccinating for clostridial diseases may decrease the occurrence of abomasal hemorrhage.[12,14] Lambs or kids on farms where such disease has been a problem can be vaccinated with multivalent bacterins against *Clostridium* infections during the first week of life.

Abomasal Impaction

Similar to rumen impaction, abomasal impaction usually occurs when poor-quality roughage is fed, but it also can be seen with foreign body obstruction of the pylorus.[4,15-17] Goats appear to be more commonly affected than sheep, and Boer goats are more commonly affected than Angora goats.[17] Pregnant animals may be more prone to this condition.

Clinical Signs and Diagnosis

Affected animals usually are anorexic. Mild distention of the ventral abdomen is characteristic, and in some cases the firm abomasum can be palpated through the abdominal wall on the right side.[18] Weight loss may be apparent. Clinicopathologic evaluation may be normal, or mild hypochloremic metabolic alkalosis may be present, with elevated rumen chloride concentrations (greater than 50 mEq/L).[18]

Treatment

Dietary changes and oral administration of mineral oil are the most commonly used treatments. Abomasotomy can be attempted, although it has rarely been reported in small ruminants and does not usually improve the long-term prognosis. For this procedure, the animal is positioned in dorsal recumbency and placed under general anesthesia. The abomasum can best be visualized through an incision parallel and to the right of midline, caudal to the xyphoid process. The prognosis is poor with or without surgery.[15]

Prevention

Dietary manipulation to improve feed or forage quality is the best mode of prevention.

Abomasal Emptying Defect

Abomasal emptying defect is a disease that manifests in similar fashion to that for abomasal impaction but is recognized only in Suffolk sheep. The underlying cause is unknown, but the proposed pathomechanism is an acquired dysautonomia from neurotoxicosis.[19] Unlike abomasal impaction, this disease is associated with concentrate feeding and often occurs around lambing time. The clinical signs are chronic weight loss, abdominal distention, and anorexia. Clinicopathologic laboratory findings and rumen chloride levels are the same as those described for abomasal impaction. At necropsy, the abomasum is greatly distended, and the contents may be liquid or dry. Treatment with laxatives, cathartics, motility modifiers, and abomasotomy has been mostly unsuccessful.[20-22]

Azalea, Laurel, and Rhododendron Toxicity

Members of the azalea, laurel, and rhododendron plant group produce andromedotoxins that alter sodium metabolism, resulting in prolonged nerve depolarization. These plants are cardiotoxic, but affected animals generally exhibit acute gastrointestinal upset. These evergreen shrubs produce thick, dark green leaves. They also have five-lobed, white to pink, saucer-shaped flowers that bloom around May-July. Some of these plants are grown as ornamental shrubbery around homes, whereas others grow wild along streams, cliffs, and rocky slopes. They can be short or tall (up to 10 m) and can form thickets. All parts of these plants are toxic.

Clinical Signs

Animals browsing a new area, those fed clippings from trimmed azalea hedges, and underfed, hungry animals with access to these plants are likely candidates for intoxication. Animals that ingest as few as two or three leaves may show salivation, tooth grinding, nasal discharge, colic, epiphora, and acute digestive upset within 6 hours of ingestion. As the intoxication progresses, animals become depressed, with a slowed pulse, and exhibit projectile vomiting and frequent defecation. Fatally intoxicated animals become paralyzed and comatose. Aspiration pneumonia secondary to the intoxication-induced impairments may develop in both sheep and goats.

Diagnosis

The diagnosis of this condition usually is based on clinical signs coupled with a history of ingestion of one of the offending plants or the discovery of such plant material in the gastrointestinal tract.

Treatment

Intoxicated animals may recover in 1 to 2 days without any therapy if the offending plants are removed from the diet. In some instances, however, the administration of charcoal (2 to 9 g/kg orally [PO]), atropine (0.05 to 0.2 mg/kg IV), other antiarrhythmic drugs, and intravenous fluids, as appropriate, may be indicated. To manage the aspiration pneumonia, antibiotics (e.g., penicillin 22,000 units/kg IM twice daily) and oral magnesium hydroxide also may be beneficial. Obviously, any existing dehydration should be corrected (see Chapter 3).

Prevention

Mountainous or hilly areas should be fenced to prevent animal access. Feeding shrubbery clippings is discouraged.

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DISEASES OF THE INTESTINES

Diarrhea in Lambs and Kids: Overview

Diarrhea in lambs and kids is a complex, multifactorial disease involving the animal's susceptibilities, the environment, nutrition, infectious agents, and management. Decades of research have been devoted to the study of the pathophysiology of infectious diarrhea in calves; the pathphysiologic picture in lambs and kids is quite similar. Despite improvements in management practices and prevention and treatment strategies, diarrhea is still the most common and costly disease affecting neonatal ruminants.[1-5]

Some general preventive measures (e.g., improved sanitation) will decrease the risk of diarrheal disease from any cause. By contrast, specific control measures such as vaccination require the definition of a specific cause of diarrhea. Table 5-2 lists the agents most likely to cause diarrhea in lambs and kids, tissues or other samples required for diagnosis, and commonly used test methods. The color and consistency of the feces and any gross lesions can appear similar with numerous diseases. Laboratory identification of infectious agents and tissue histopathologic examination are therefore key to establishing a diagnosis (see Chapters 16 and 20). Because autolysis and secondary bacterial invasion of the gut begin within minutes of death, necropsy samples taken immediately from euthanized lambs and kids yield the most reliable diagnostic material. Mixed infections with two or more pathogens are common, and clinically important farm-specific pathogens change from year to year.[3-7] In some cases an underlying nutritional deficiency or excess may be present concurrently with infective disease. The clinician should therefore take a variety of samples to ensure identification of

TABLE 5-2 Diagnostic Samples and Testing Methods Required for Differentiation of the Most Common Causes of Infectious Diarrhea in Lambs and Kids

Causative Agent	Sample Required	Test Method*
Escherichia coli	2 to 3 g feces	Culture and serotyping for K99 and F41
	Formalin-fixed small intestine	Histopathologic examination
Rotavirus	2 to 3 g feces or colonic contents	EM, ELISA, VI, CF test, PCR assay
	Formalin-fixed small and large intestine	Histopathologic examination
	Frozen small and large intestine	VI, FA test, IP assay
Cryptosporidia	2 to 3 g feces	FA test, fecal flotation
	Air-dried fecal smear	Acid-fast stain
	Formalin-fixed small and large intestine	Histopathologic examination
Salmonella	2 to 3 g feces	Culture, PCR assay
	Formalin-fixed small and large intestine	Histopathologic examination
	Frozen small and large intestine and mesenteric lymph nodes	Culture
Giardia	Wet mount of feces	Iodine staining
	Feces	ELISA, FA test
Clostridium perfringens	Frozen small intestinal contents and abomasum, small and large intestine	Culture, toxin identification
	Formalin-fixed abomasum and small and large intestine	Histopathologic examination
Coccidia	2 to 3 g feces	Fecal flotation
	Formalin-fixed small and large intestine	Histopathologic examination

CF, *Complement fixation;* ELISA, *enzyme-linked immunospecific assay;* EM, *electron microscopy;* FA, *fluorescent antibody;* IP, *immunoperoxidase;* PCR, *polymerase chain reaction;* VI, *virus isolation.*
Data from Rings DM, Rings MB: Managing Cryptosporidium *and* Giardia *infections in domestic ruminants,* Vet Med *91:1125, 1996; Cohen ND, et al: Comparison of polymerase chain reaction and microbiological culture for detection of salmonella in equine feces and environmental samples,* Am J Vet Res *57:780, 1996; and Drolet R, Fairbrother JM, Vaillancourt D: Attaching and effacing* Escherichia coli *in a goat with diarrhea,* Can Vet J *35:122, 1994.*

all pathogens and predisposing factors involved; continued reevaluation of the causes of diarrhea is crucial. Evaluation of material from multiple cases, with a focus on those in the acute phases, is important. Although examination of antemortem fecal samples can be diagnostic, laboratory testing of tissue samples may yield better results.

Treatment and preventive measures for specific diarrheal diseases are the focus of the remainder of this section, which is followed by sections on general supportive treatment and control measures for all infectious diarrheal diseases.

Causes of Diarrhea in Neonatal Lambs and Kids

Four major pathogens cause diarrhea in lambs and kids during the first month of life: enterotoxigenic *Escherichia coli* (ETEC), rotavirus, *Cryptosporidium* species, and *Salmonella* species. The relative prevalence of these infectious agents varies greatly among studies. This variability probably results from differences in location, season, and diagnostic techniques and the occurrence of mixed infections. Other, less common causes of diarrhea in neonates are *Giardia* infections and nutritional diarrhea.

Enterotoxigenic *Escherichia coli*

Pathogenesis

ETEC employs two virulence factors to cause disease. The first is the ability to attach and colonize the intestinal villi, which is accomplished by means of fimbriae or pili. The most important fimbriae in lambs are K99 and F41.[8] The fimbrial antigens can be recognized from samples sent for analysis in most diagnostic laboratories and are important in identifying this agent as a cause of diarrhea. After the organism attaches to the villi, it produces the second virulence factor, enterotoxin. Enterotoxin interferes with the normal physiology of the gut, with resultant diarrhea.[8] Calves have an age-associated resistance that probably is related to the blocking of fimbrial attachment to the gut, so ETEC diarrheal disease occurs mainly in calves younger than 1 week of age.[9,10] The mode of infection is fecal-oral.

Clinical Signs

ETEC diarrhea is seen in lambs and kids younger than 10 days of age but is most common at 1 to 4 days, so age-related resistance also may be a factor in newborns of these species.[3,7] It usually manifests as an outbreak in lambs and kids between 12 and 48 hours of age. Because ETEC causes a secretory-type diarrhea, bicarbonate loss in the diarrhea leads to severe acidosis, with lambs and kids quickly becoming dehydrated and recumbent. However, many infected animals die before developing diarrhea. Affected neonates are depressed, stop nursing, and may show excessive salivation. Fluid sequestration in the abomasum produces a splashing sound on movement. This condition is associated with high mortality if animals are not treated promptly.[7,8]

Diagnosis

Fecal culture and serotyping for the K99 and F41 fimbrial antigens constitute the basis for diagnosis. Because many nonpathogenic *E. coli* bacteria are normal gut inhabitants, growth of this organism on cultures usually is an insignificant finding.[8] Occasionally the bacteria do not express the fimbrial antigens in culture, so ETEC cannot be ruled out if the culture is negative for K99 and F41.[11] Histopathologic evidence of colonization of the small intestine can support a diagnosis.

Treatment

Supportive care consisting of fluid therapy with either oral, intravenous, or subcutaneous administration of a polyionic solution is the mainstay of therapy. The use of oral antimicrobial agents is controversial. Although antibiotics may kill the ETEC, they also may interfere with normal gut flora. If fluid support is provided, the diarrhea usually subsides without antibiotic treatment. Nevertheless, oral neomycin (10 to 22 mg/kg twice daily) or trimethoprim-sulfa (30 mg/kg PO) and systemic ampicillin (10 to 20 mg/kg IM twice a day) or amoxicillin (10 to 20 mg/kg IM three times a day) may be beneficial. NSAIDs are indicated to decrease inflammation of the gut and provide some analgesia. The use of flunixin meglumine (1 to 2 mg/kg IM) has been shown to decrease fecal output in ETEC infections in calves[12] and appears to be beneficial in lambs.

Prevention

Vaccination of ewes and does with bovine ETEC vaccine before they give birth is recommended to increase passive immunity in the neonate.[3,4,8] Monoclonal and polyclonal antibody products for calves may be beneficial during an outbreak if administered to lambs or kids within the first 12 hours of life. The use of neomycin (10 to 12 mg/kg PO twice daily) in lambs that appear clinically normal may help stop the progression of an outbreak. Shearing ewes pre-partum to minimize fecal ingestion by neonates and ensuring that newborns ingest adequate colostrum both will help decrease the incidence of this disease. Making sure that ewes and does have a 2.5 to 3.5 body condition score at parturition and are fed adequate diets in the final 2 months of gestation will increase the chance of adequate colostrum manufacture by the dam.

Rotavirus

Pathogenesis

Lambs and kids are infected with group B rotaviruses, whereas most other animals and human beings are infected with group A rotaviruses.[13] Rotaviruses infect villus tip cells of the small intestine, which results in villus atrophy and malabsorptive diarrhea.[14]

Clinical Signs

Rotavirus generally causes diarrhea in lambs and kids 2 to 14 days of age, but older animals also can be affected. Young animals can become very depressed and dehydrated.[3,13,15,16]

Diagnosis

Detection of the organism by electron microscopy of fecal or colonic samples or by immunologic techniques applied to feces or tissue sections is the basis of diagnosis.[13,16] Because these organisms are sloughed with the villus tip cells they infect, and viral antigens are complexed with the animal's antibodies, tissue samples from acutely infected animals are of highest diagnostic value.[17] Rotavirus has been detected in animals without diarrhea, so other causes of diarrhea should be investigated as well.[4,6]

Treatment and Prevention

Rotavirus diarrhea is treated with supportive care. Prevention by vaccination of ewes and does with bovine rotavirus vaccines before they give birth is recommended to increase passive immunity in neonates.[3,4]

Cryptosporidium Species

Pathogenesis

Cryptosporidium parvum is a protozoan that can cause a malabsorptive diarrhea similar to that seen with rotavirus infection. Unlike other protozoal agents, such as the one that causes coccidiosis, cryptosporidia do not require fecal excretion for sporulation to infective stages.[18] They sporulate in the gut, whereupon approximately 20% become immediately infectious to other villus tip cells without leaving the intestines. This method of autoinfection can result in severe disease that may be sustained for long periods. Because some of the oocysts also are immediately infectious when they are shed in feces, spread of infection may be rapid.

Clinical Signs

Cryptosporidia can cause diarrhea in lambs and kids at 5 to 10 days of age.[4,19,20] Affected animals often are active, alert, and nursing. The diarrheal stools usually are very liquid and yellow. Diarrhea can range from mild and self-limiting to severe, especially with mixed infections.[4,6,19,21] Relapses are quite common, and this organism usually occurs as a component of mixed infections.

Diagnosis

Acid-fast staining of air-dried fecal smears is a quick and easy method of diagnosis. Examination under 40× to 100× magnification reveals round protozoa that have taken up the red color of the carbol fuchsin portions of the stain on a green background (Figure 5-12). Although cryptosporidial infection can be diagnosed by fecal flotation testing, the very small size (4 to 6 μm) of these organisms makes this method difficult and subject to false-negative results.[22,23] Both immunologic and polymerase chain reaction (PCR) techniques have been developed to improve detection limits.[22,24] Cryptosporidia also can be identified on histopathologic examination. Cryptosporidiosis is a zoonotic disease, and people can become infected from handling infected animals or feces.[18]

Prevention

No consistently effective treatment for cryptosporidiosis in ruminants has been identified. However, proper hydration and electrolyte balance should be maintained, along with other supportive care. Prevention through decreased exposure of lambs and kids to organisms in the environment is critical, especially exposure of neonates at birth.[25] On farms endemic with coccidiosis or during an outbreak, improved hygiene may be of benefit (e.g., pre–colostral intake udder wash, feeding only low-heat-pasteurized colostrums, isolation of all exposed animals). Anecdotal reports suggest that decoquinate and monensin sodium may be useful in control of cryptosporosis. Decoquinate (2.5 mg/kg PO) fed to does and kids may be useful in decreasing morbidity and mortality associated with cryptospirosis in goat kids.[26] Treatment in all affected animals also should include fluid-electrolyte therapy.

During an outbreak, affected animals should be isolated from the rest of the flock. No new animals should be added to a pen in which the disease has been diagnosed. Keepers should depopulate pens in which the disease has been diagnosed and attempt to clean the environment. Cryptosporidiosis can be particularly difficult to control because of the organism's persistence in the environment and resistance to most chemical disinfectants. Ammonia (5% to 10%) and formalin (10%) seem to be the most effective agents, but due to potential for toxic effects caution is indicated with the use of either chemical.[19,27] Feeders should be constructed to minimize fecal contamination. Early results are favorable for vaccine development in cattle, and vaccination may prove to be the best control method in the future.[28] Cryptosporidiosis is potentially a zoonotic disease; clinicians and keepers should therefore exercise great caution when handling affected animals, and well-planned biosecurity programs should be instituted (see Chapter 19).

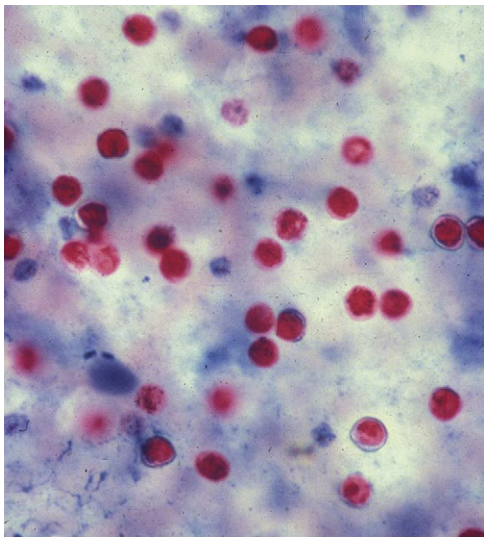

Figure 5-12 Red-staining *Cryptosporidium* on a blue-green background in a fecal smear prepared with an acid-fast stain. This protozoal parasite induces villus atrophy and decreased digestion.

Salmonella Species

Pathogenesis

The bacterial genus *Salmonella* has thousands of serotypes, all of which can potentially cause diarrhea in animals. Salmonellae can cause diarrhea in lambs and kids of any age.[3,4] These microbes produce enterotoxins, are invasive, and cause severe inflammatory disease and necrosis of the lining of the small and large intestines.

Clinical Signs

Affected animals younger than 1 week of age are more likely to die acutely before onset of clinical signs, whereas animals older than 1 week are more likely to have diarrhea.[4,7,29] An acute onset of fever, depression, tenesmus, and shock occasionally is observed. *Salmonella*-induced diarrheal stool is more likely to contain blood.[4] Enteric salmonellosis also is a zoonotic disease that warrants implementation of protective measures.

Diagnosis

A diagnosis of *Salmonella* diarrhea is based on culture of the organism in feces or tissues and characteristic changes on histopathologic examination of the small

and large intestine.[30] More sensitive PCR techniques for identifying *Salmonella* species in feces are being developed.[31] The diarrheal feces occasionally may contain fibrin, but many animals die before this development is observed. The clinician may note leukopenia or leukocytosis in the CBC results.

Treatment

Therapy for *Salmonella*-induced diarrhea involves supportive care and possibly parenteral antimicrobial therapy. The use of antimicrobial agents is controversial and probably does not influence the gastrointestinal infection. Nevertheless, because *Salmonella* is an invasive organism, parenteral use of antimicrobial agents may be beneficial in preventing septicemia. Antimicrobial susceptibility patterns are difficult to predict for *Salmonella* species, so antimicrobial therapy should be based on culture and sensitivity results. Ceftiofur sodium (1.1 to 2.2 mg/kg IM twice daily) or trimethoprim-sulfadiazine (15 mg/kg SC once a day) can be administered until antimicrobial sensitivity results are available.

Prevention

Latent carriers of *Salmonella* can potentially shed organisms to other animals, particularly when they are stressed.[4] Newly introduced animals should be isolated for 1 month, and fecal culture should be considered.[4] Bleach (sodium hypochlorite) and chlorhexidine are effective disinfectants to apply to the premises and animal handling/feeding equipment during an outbreak. Identification of carrier animals by fecal culture is recommended for herd problems. Vaccine efficacy is questionable, and to date its effects have not been thoroughly evaluated in sheep and goats.[32]

Giardia

Giardia-induced diarrhea is more commonly seen in, but not limited to, 2- to 4-week-old lambs and kids.[4,33] The diarrhea usually is transient, but infected animals can continue to shed cysts for many weeks, even when they appear to be clinically normal.[22,34,35] Therefore simply finding the pathogen in feces does not mean that it is the cause of the diarrhea, especially in older animals. *Giardia* can be found in herds without any history of neonatal diarrhea, so finding *Giardia* in herds in which newborn animals are experiencing diarrhea is of questionable relevance.[36] However, these animals may be a source of infection for others and possibly humans.[22,33] Identification of the organism on iodine-stained wet mounts of feces or tissue is the classic method of diagnosing giardiasis, but more sensitive immunologic techniques are now available.[22,33] Infected animals can be treated effectively with fenbendazole (5 to 10 mg/kg twice daily for 3 days or once daily for 5 days).[22] Giardiasis has historically been treated with oral metronidazole (50 mg/kg once a day for 5 days). However, use of this drug class in food animals is currently illegal in the United States. Giardiasis is potentially a zoonotic condition.

Nutritional Diarrhea

Infectious agents are not the only cause of diarrhea in neonates. Nutritional problems can result in diarrhea, but cases related to nutrition are underreported in the literature because the resulting diarrhea usually is mild and subsides without treatment. Nutritional diarrhea is most common in orphaned animals and usually is a result of improper management practices such as use of poor-quality milk replacers, mixing errors, or infrequent feeding of large amounts (see Chapter 2). Diarrhea resulting from consumption of lush pasture or high-energy rations also is commonly seen and usually is self-limiting. The incidence of this form of gastric upset can be minimized by a slow introduction (over 2 to 3 weeks) to energy-dense diets.

In calves with infectious diarrhea that develop maldigestion or malabsorption, secondary nutritional diarrhea may result from an inability to digest carbohydrates (lactose, xylose).[37,38] This digestive defect has been reported in goats and also is probably a cause of diarrhea in lambs.[39] Diarrhea resulting from primary lactose deficiency also has been reported in calves.[40] Calves on poor-quality milk replacers can develop an overgrowth of normal enteric *E. coli*, resulting in diarrhea.[41] If lactose intolerance is suspected, decreasing the amount of lactose fed and using commercially available lactose enzymes may alleviate clinical problems.

Causes of Diarrhea in Older Lambs and Kids

The most common cause of diarrhea in older lambs and kids is nematode infestation. Other major causes of diarrhea in older lambs and kids are *C. perfringens* infection and coccidiosis. Coccidiosis is covered in Chapter 6. *Giardia* has been reported to cause weight loss without diarrhea in 2- to 3-month-old lambs.[42]

Clostridium perfringens

C. perfringens types A, B, C, and D all can cause diarrhea in lambs and kids, but type D is the most common etiologic agent in the United States.[4,7,43,44]

Pathogenesis

Clostridial diarrhea occurs in peracute, acute, and chronic forms and commonly is called *enterotoxemia* or *overeating disease.* In type C infection, a beta toxin can cause acute hemorrhagic enteritis. Type C infection is seen mostly in lambs or kids younger than 3 weeks of

age. An epsilon toxin is responsible for pathologic findings in type D infections. Enterotoxemia usually is seen in rapidly growing feedlot lambs on high-concentrate rations. It also is associated with other feeding changes, including changes in type of pasture. However, it occasionally has been reported in the absence of any dietary changes, particularly in goats.[4,7,45] This disease commonly occurs in the fastest-growing and most well-conditioned animals. Even vaccinated herds (again, more usually goats) can be affected, so it should not be ruled out despite confirmation of previous vaccination.[4]

Clinical Signs

The *peracute* form of clostridial infection is characterized by the rapid onset of severe depression, abdominal pain, profuse and bloody diarrhea, and neurologic signs. Death occurs within hours of onset of clinical manifestations. Sudden death may occur without diarrhea. Sudden death following the onset of neurologic signs is more common in sheep, whereas goats are more likely to show signs of diarrhea before death.[4] Similar but less severe signs are seen in the *acute* form of the disease. The *chronic* form occurs more commonly in goats.[4,44]

Diagnosis

Antemortem diagnosis is based on clinical signs. At necropsy, *C. perfringens* can be cultured from intestinal tissue samples. The significance of a positive culture can be difficult to interpret, however, because these organisms can be a normal component of the gut flora but subsequently proliferate after death. This is true especially of type A, for which a role in actual disease is controversial.[46] Histopathologic examination of sections of the gut can be helpful. Identification of the toxins (namely, the epsilon toxin) in intestinal contents is required for a definitive diagnosis.[4,7] Because the toxin degrades within several hours of death, its absence does not preclude enterotoxemia as a diagnosis.[43]

Treatment

Treatment is rarely effective but consists mainly of aggressive supportive care. *C. perfringens* type D antitoxins (15 to 20 mL SC) can be administered to animals during an outbreak of enterotoxemia if clinical signs are noted. The antitoxin may be more effectively used as a preventive early in an outbreak of the disease. During an outbreak, any animals that have not been vaccinated should be given the antitoxin and vaccinated with the toxoid simultaneously; those previously vaccinated should receive a booster vaccination.

Prevention

Routine vaccination should start at 4 to 6 weeks of age and be followed by a booster 3 to 4 weeks later. In settings in which the disease has become endemic, however, lambs or kids can be vaccinated and given antitoxin during the first week of life. Yearly vaccination, preferably a few weeks before the ewes and dams give birth, increases colostral immunity in neonates and improves prevention programs. Goats may not respond as well to vaccination as sheep do, so biannual, triannual, or quarterly vaccination is recommended, especially in herds in disease-endemic areas.[4,39] Vaccination with only *C. perfringens* type C and type D vaccines and tetanus toxoid is superior to the use of more polyvalent clostridial vaccines.[4] Reducing the energy density of the diet and avoiding sudden dietary changes or alterations in the feeding routine are crucial to prevention. Control of internal parasites, particularly tapeworms, may further reduce the incidence of these disorders.

Miscellaneous Causes of Diarrhea in Kids and Lambs

Adenovirus, caprine herpesvirus, coronavirus, *Campylobacter jejuni*, *Escherichia fergusonii*, *Yersinia* species, and *Strongyloides papillosus* can cause diarrhea in lambs and kids of various ages.[2,4,6] Enterohemorrhagic *E. coli* (EHEC) and enteropathogenic *E. coli* (EPEC) also have been isolated from feces of both diarrheic and normal lambs and kids.[48-52] These *E. coli* serotypes are K99- and F41-negative. Culture and serotyping of these organisms from feces and tissue samples with typical histopathologic lesions are diagnostic. Although ETEC disease is not zoonotic, EHEC and EPEC can potentially affect humans and cause food-borne illness.

TREATMENT OF LAMBS AND KIDS WITH DIARRHEA

Although specific therapies are available for some causes of diarrhea, many animals need to be treated for dehydration and metabolic acidosis regardless of the inciting cause. Animals with only mild diarrhea, especially mild nutritional diarrhea, may not require therapy unless they become dehydrated. If kids or lambs become less than 8% dehydrated and are only mildly depressed but still willing to nurse, they can be treated with oral electrolytes designed for calves. Fluids can be administered by bottle or by feeding tube (~ 18-24 inch, 3/8 inch diameter, catheter tip) if the animal will not nurse. The keeper or the clinician should carefully adjust the amount of fluids for lambs and kids (250 to 500 mL [8 to 16 oz], as opposed to 4 L in a calf). Because most electrolyte solutions designed for calves contain glucose, they should be refrigerated after they have been mixed and any leftovers discarded within 24 hours. Intravenous fluids may be needed to treat more severe dehydration. If the lamb or kid is too weak to stand, intravenous fluids are indicated. Isotonic fluids containing electrolytes should be given to

replenish losses. Glucose can be added to make a 1% to 2.5% solution. Sodium bicarbonate also may be administered, especially if the dehydration is severe. A rule of thumb is to give one fourth of the calculated fluid needed as isotonic bicarbonate (1.3%). Extra potassium (10 to 20 mEq/L) can be added to fluids, because most animals are severely dehydrated from diarrhea and depleted in potassium, even though their blood potassium levels may be elevated. If extra potassium is added, acidosis must be corrected concurrently. After correcting the dehydration, the keeper or the clinician can offer oral electrolyte-enriched fluids to replace ongoing losses caused by continued diarrhea (see also Chapter 3).

Removing milk or milk replacer from the diet is not recommended. Young animals need nutrients, and even high-energy, glucose-containing electrolyte solutions are no substitute for milk. Animals should continue to receive milk replacer in normal amounts or be allowed to nurse; oral electrolytes also can be given if necessary. Animals being hand-fed should be offered small amounts frequently to help minimize problems. Electrolytes should never be mixed with milk but should instead be given in separate feedings. If lactase deficiency is suspected, lactase drops or capsules (available in health food stores) can be added to milk or milk replacer.[39]

NSAIDs (e.g., flunixin meglumine, 1.1 to 2.2 mg/kg IV, or ketoprofen, 3 mg/kg IV) are beneficial, especially if toxemia is involved, as in ETEC, enterotoxemia, and salmonellosis. Antimicrobial agents should be reserved for proven outbreaks of salmonellosis and for animals with other causes of diarrhea that do not respond to fluid therapy and NSAIDs; these drugs should be administered only parenterally. Oral coating agents and antacids are popular, but such agents have not been shown to be beneficial, and their use is not therapeutically logical in light of the pathogenesis of these diseases. The therapeutic use of probiotics is questionable, but anecdotal reports suggest they may be beneficial in reestablishing the normal flora of the small intestine. Our own rule of thumb is that nothing should be given orally except milk, oral electrolytes, and possibly probiotics.

GENERAL CONTROL MEASURES FOR INFECTIOUS DIARRHEA

Ensuring adequate intake of high-quality colostrum and minimizing stress are important for prevention of all neonatal diseases. A normal lamb or kid will stand and nurse within 45 minutes to 1 hour of birth. The ingestion of colostrum within 2 to 3 hours is essential in preventing hypothermia and hypoglycemia and decreasing the incidence of various diseases. Lambs or kids born as twins or triplets, weak or injured neonates, those born during severe weather, those born from a dam with dystocia, and those delivered by cesarean section all are candidates for colostrum supplementation. Supplemental colostrum should be good-quality colostrum from females that have tested negative for Johne's disease, ovine progressive pneumonia (OPP), and caprine arthritis encephalitis (CAE). Mixing colostrum from several cows decreases the incidence of the "cow colostrum–associated" hemolytic disease sometimes seen in lambs. If the lamb or kid is unable to nurse, it should be tube fed 50 mL/kg of colostrum. The veterinarian or animal handler can sit comfortably holding the lamb or kid in sternal recumbency in the lap. A 12 to 14 French soft feeding tube is then lubricated, inserted into the side of the mouth, and passed slowly to the depth of the thoracic inlet. If the tube is placed in the trachea, the lamb or kid will show signs of discomfort and may shake and cough. The tube may be palpated on the left side of the throat. After correct placement of the tube, colostrum can be administered by gravity flow.

Antepartum shearing of the dam may decrease the likelihood of ingestion of feces by lambs. Good sanitation in lambing and kidding areas is paramount in management programs that stress prevention. The presence of organic matter interferes with the effectiveness of many disinfectants, so removal and proper disposal of feces, carcasses, and placentas are essential. When disposing of waste material containing either *Cryptosporidium* or *Giardia,* the keeper should be careful to avoid contaminating water sources. Infected animals should be isolated to prevent spread of the infection throughout the flock or herd. In general, infected animals should remain in the environment where the infection was first diagnosed, because it is already contaminated. Removing pregnant ewes or dams to a clean area before lambing or kidding helps minimize the continued spread of disease. If possible, lambs and kids already born but not showing clinical signs should be removed to a third area. If "safe" pastures are maintained for internal nematode control, they are ideal for use in an emergency situation to control these diseases (see also Chapter 6). Although some animals may appear normal, they may be incubating and possibly shedding the infective agents of disease. If such animals are moved with pregnant females, they can be a source of contamination in a clean area. If healthy lambs and kids cannot be moved to a third, relatively safe area, they should be left with the clinically infected animals because they have already been exposed.

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DIARRHEA IN ADULT SHEEP AND GOATS

The list of considerations in the differential diagnosis for acute and chronic diarrhea in small ruminants is extensive.[1,2] The most common cause of diarrhea in adult sheep and goats is parasitism; another major cause is Johne's disease. Parasitism is discussed in Chapter 6. Other causes of acute diarrhea include rumen acidosis, peritonitis, endotoxemia, and ingestion of toxins. The list of toxins that cause diarrhea also is very long, and often diarrhea is not the primary clinical sign. Some of the more commonly encountered toxins that produce diarrhea are arsenic, salt in toxic amounts, levamisole,

copper, oak, selenium, and pyrrolizidine alkaloids.[1] *Salmonella* infection and chronic enterotoxemia can cause diarrhea in adult animals. Coccidiosis can occur in adults under severe stress or in animals that possess limited immunity because of lack of exposure. Hepatic and renal disease and copper deficiency sometimes are accompanied by chronic diarrhea, but weight loss is a more common sign in adults.

Johne's Disease

Johne's disease, also called *paratuberculosis*, is a chronic wasting and diarrheal disease caused by the bacterium *Mycobacterium avium* subspecies *paratuberculosis.* Transmission of the organism is primarily by the fecal-oral route. Young animals are more susceptible to infection than adults. It can be transmitted through milk and placenta.

Pathogenesis

Bacterial shedding in feces and milk and transplacental transmission are more common in animals showing clinical signs.[3-5] Therefore the offspring of infected animals, and especially the offspring of animals showing clinical signs, are more likely to acquire the infection than other members of the flock/herd. After an animal is exposed, it will either clear the organism or acquire a chronic, persistent infection. The infection most commonly is isolated to the ileal regions of the small intestine, where it causes granulomatous thickening of the intestinal wall and subsequent malabsorptive diarrhea. Infected animals may be asymptomatic for years.

Clinical Signs

Morbidity rates are low (approximately 5%), but for every infected animal with clinical signs, several are in the subclinical state and may be a source of both horizontal and vertical transmission.[3] Both sheep and goats appear to remain asymptomatic until they reach 2 to 7 years of age. The most consistent clinical sign in sheep and goats is chronic weight loss. Chronic diarrhea occurs in approximately 20% of cases.[3] Signs may appear with or be exacerbated by stress, especially after parturition.[3,4] Hypoproteinemia and chronic mild anemia are the only consistent findings from clinicopathologic laboratory tests. Submandibular edema may develop as a consequence of low protein levels in infected animals, and because parasitism is ubiquitous, an accurate diagnosis may be difficult.

Diagnosis

Diagnosis is by culture of the organism from feces. Such testing unfortunately takes between 8 and 14 weeks but can identify 40% to 60% of clinically infected goats. Feces of noninfected sheep and goats within heavily infected herds can yield a positive culture from oral-fecal pass-through of the organism. Sheep strains of Johne's disease and some goat variant strains seem to be more difficult to culture in media used to identify cattle strains of the disease. Therefore fecal culture in sheep and goats appears to be of limited benefit in a clinical setting.[4,5] A relatively inexpensive and easily performed method of identifying approximately 50% of all clinically infected animals is acid-fast staining of fecal smears.[3,4] A PCR fecal assay also is available, but its sensitivity is lower than that of fecal culture. Good diagnostic results can be obtained with serologic testing for antibodies (e.g., agar gel immunodiffusion [AGID] test, enzyme-linked immunospecific assay [ELISA], complement fixation test) in animals showing clinical signs. The specificity of all of the serologic tests is greater than 95% in sheep and goats with signs of clinical disease, although the sensitivity is not as high.[4-7] Therefore a positive serologic test result in an animal showing clinical signs indicates that the animal has Johne's disease. However, the disease cannot be ruled out with a negative test result. Identification of subclinically infected animals using serologic tests is more problematic. A sensitivity of approximately 50% is all that should be expected. With the ELISA and complement fixation test, cross-reactivity with *Corynebacterium pseudotuberculosis* may occur, thereby limiting the value of such testing in flocks with caseous lymphadenitis infections.[4,8] ELISA performed on milk samples from goats had reduced sensitivity but increased specificity (less cross-reaction) compared with serum ELISA.[9] Sheep and goats appear to respond differently with regard to the formation of antibodies. In sheep, antibodies tend to develop in the later stages of the disease, whereas antibodies may be detected much earlier in the goat. Necropsy diagnosis is based on the finding of thickened, corrugated intestines, especially in the area of the ileum. Acid-fast staining of impression smears (taken from the ileum and ileocecal lymph nodes) can help yield a quick diagnosis. The staining of numerous clumps of acid-fast rods is highly suggestive of Johne's disease.

Prevention

Johne's disease has no effective treatment, so prevention and control are imperative. However, preventing the introduction of Johne's disease into a herd can be difficult. Because animals with subclinical infection may not shed the organism or shedding may occur in only small quantities, fecal culture is helpful only if a positive culture is obtained. The sensitivity of serologic tests of animals with subclinical disease is low and variable among flocks.[4,5] Negative test results in subclinically infected animals are common. However, the specificity of serologic tests remains high, so a positive test result is a valid reason to not purchase an animal.[4] Because Johne's disease also occurs in cattle, supplemental colostrum supplies should come only from dairy herds free of Johne's disease.

After Johne's disease is diagnosed in a herd, several control measures should be implemented. Sanitation is important, because the organism is highly resistant in the environment (i.e., capable of surviving longer than 1 year under most conditions).[5] Reduced stocking rates, frequent cleaning of pens, and use of automatic waterers will decrease fecal transmission. Keepers and herdsmen should cull the offspring of infected animals. Culling animals on the basis of the results of flock/herd -wide AGID testing or ELISA and fecal culture is recommended. Animals should be tested at least once a year. More frequent testing as resources allow will speed the identification of infected animals. A vaccine for cattle is available only in some locales, and clinicians or keepers may require official permission for its (extralabel) use in sheep and goats. Vaccination for Johne's disease in cattle does not eliminate infection but can decrease herd prevalence, delay the onset of clinical signs, and decrease cross-transmission by infective bacterial shedding in the feces.

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INTESTINAL OBSTRUCTION

Any cause of intestinal obstruction that occurs in other ruminants may occur in sheep and goats. Most of these diseases produce abdominal discomfort and occasionally abdominal distention. Diagnosis is made more difficult owing to size restrictions that usually preclude rectal palpation. Abdominal radiographs and ultrasonography may help differentiate among these diseases, but exploratory surgery or laparoscopic evaluation may be required to obtain a definitive diagnosis and to permit selection of appropriate therapeutic plans.

Intussusception

Although occurring in all ages, intussusception is more common in young animals. In this condition, one segment of the intestine telescopes into an adjacent segment. Any portion of the intestine can be affected, but the ileum and ileocecal junction are the most common areas involved. When intussusception occurs, the intestinal lumen narrows to the point of obstruction. The initiating cause is not always known.[1,2] The condition is associated with an intestinal mass in adults and enteritis in young animals.[1] *Oesophagostomum* infestations have been implicated as a cause in sheep.[1]

Clinical Signs

The initial complaint is colic (manifested as kicking at the abdomen, repeated rising and lying down, and vocalization), typically followed by chronic low-grade pain. True colic signs are variable in lambs and kids. In some instances, after the initial colic episode subsides, animals show no evidence of pain until the abdomen becomes distended. The time between the initial intussusception and abdominal distention depends on where the blockage occurs. With intussusception of the ileum, several days may elapse before bilaterally symmetric abdominal distention becomes evident. Fecal output is scant, and what little there is may be dark or tarry or may contain mucus. Dehydration becomes evident, hypochloremic metabolic alkalosis may develop, and rumen chloride levels may increase with obstructions of the duodenum.

Diagnosis

Abdominocentesis may yield fluid compatible with a transudate (increased protein concentration and leukocyte numbers).[1] Plain radiography and ultrasonography may reveal fluid-distended intestinal loops. Occasionally the intussusception itself can be visualized with ultrasonography or palpable through the abdominal wall. If the condition is not treated, intestinal rupture and peritonitis can occur.

Treatment

Surgical correction is required. If the intussusception is corrected early, the prognosis is good in the absence of peritonitis. Fluid support is needed to correct dehydration and metabolic abnormalities. Intravenous fluids should be administered until rumen function returns. Ringer's injection with added calcium (approximately 25 mL of calcium borogluconate/L) and potassium (10 to 20 mEq/L) are good choices for fluid therapy.

Foreign Body Obstruction

Ingested foreign bodies or bezoars can obstruct portions of the intestines.[3,4] The signs are similar to those of obstruction caused by intussusception and depend on which part of the intestine is blocked. In some cases the obstructing body can be seen with use of radiography or ultrasonography. Surgical removal is required for treatment.

Cecal Volvulus and Torsion of the Root of the Mesentery

Cecal volvulus and torsion of the root of the mesentery occur sporadically in sheep and goats.[1,3] Signs of extreme abdominal pain, rapidly progressive abdominal distention, and circulatory collapse are characteristic clinical manifestations. Immediate surgical correction and circulatory support are needed.

Intestinal Atresia

Atresia of the colon, rectum, or anus can occur as a congenital problem. The clinical sign of progressive abdominal distention usually is noted in the first week of life. Atresia of the anus can be detected on physical examination, but atresia of the colon and rectum may require contrast radiography for definitive diagnosis. Surgical establishment of anal patency can be performed for atresia ani. A permanent colostomy may be required in animals with atresia of the colon and rectum. Atresia of the anus and rectum are considered heritable in cattle.[1] In our experience, atresia ani is more common in sheep than in goats.

If *surgical correction* of atresia ani is attempted, the animal also should be neutered at this time, because of the potential genetic basis for this condition. Although correction can be done with local anesthetic infiltration, we recommend epidural anesthesia in case the correction is not as simple as expected. General anesthesia should be considered for cases that will require deeper dissection to pull rectal tissue to the normal anus location (see Chapter 18). Ultrasonography can be used to locate a feces-filled rectum before the surgery is begun. Occasionally a slight bulge in the skin may be seen where the anus should be located. This finding is more common in males. In females with atresia ani, rectovaginal fistula with defecation through the vagina is a common finding. As a result, females may not present as early in the course of the condition as males. The owner or manager may choose to allow affected females to live until slaughter weight is reached, because surgical correction is difficult and therefore not economically feasible for a market animal. Additionally, attempts at surgical correction at more advanced stages of the condition are seldom successful. An important consideration is that over time, the fistula may not remain large enough to allow normal defecation. Therefore constipation with subsequent rectal dilation may occur before the female reaches market weight.

The best candidates for corrective surgery are animals that exhibit the aforementioned bulge, whether male or female, without a rectovaginal fistula. This bulge in the skin indicates the rectum is most likely immediately cranial to the imperforate skin. For surgical correction, the area around the anus is clipped if needed and scrubbed for surgery. Epidural or local anesthesia is obtained. The clinician should first make a stab incision through the imperforate skin to identify the rectum. When feces are seen to freely exit this incision, it can be converted to an X-shaped incision; in such cases, a patent anus will be maintained by expulsion of feces. Some surgeons prefer to make a circular incision, which will be less likely to heal together, once again obstructing defecation. With this circular incision, repair is best accomplished by securing the rectal mucosa to the edge of the incised skin with absorbable suture placed at four points equidistant around the circular skin incision. The space between these sutures can then be closed with either short segments of continuous suture or additional separate sutures. The mucosa-to-skin suture procedure is not very difficult in a tractable patient with use of appropriate regional anesthesia. Use of this closure technique is imperative if the rectum terminates further cranial to the skin and some dissection with caudal traction on the rectum is required. Postoperatively, the animal should be given mineral oil, DSS, or stool softeners as indicated.

Intestinal Ileus

Ileus of the small intestine is a pseudoobstruction resulting from the absence of intestinal motility. The animal's failure to pass ingesta leads to signs similar to those of intussusception. The cause of ileus usually is unclear, but the condition often is secondary to systemic disease. The same elements that cause rumen stasis may potentially result in intestinal stasis and ileus. Symptomatic treatment with NSAIDs for pain and inflammation and fluids for dehydration is usually curative.[1] If signs persist, however, surgical exploration is indicated to rule out true obstructive diseases.

Peritonitis

Pathogenesis

Infection of the peritoneal lining of the abdominal cavity may lead to septic peritonitis. Common causes include uterine tears; rupture of the rumen or abomasum secondary to rumenitis, abomasitis, or abomasal ulcers; trocarization of the rumen for bloat; and rupture of the intestine secondary to obstruction.

Clinical Signs

Signs of peritonitis depend on the extent and severity of the underlying condition. Abdominal discomfort and distention, dehydration, injected mucous membranes, depression, and death all can occur. The presence of a fever is variable, both heart rate and respiratory rate usually are elevated, and respiratory effort may be guarded. Animals may be febrile early but exhibit a normal to low body temperature as the condition progresses.

Diagnosis

Abdominal ultrasound imaging can be useful in locating pockets of fluid for abdominocentesis, which usually yields fluid with increased protein concentration and leukocyte numbers. On occasion, intracellular bacteria are observed on cytologic examination. The presence of extracellular bacteria is not diagnostic, because accidental enterocentesis can occur. Culture of abdominal fluid with antimicrobial sensitivity testing is indicated for proper treatment. The causative organisms vary depending on the source of the bacteria. Rumen bacteria typically are gram-negative anaerobes; *E. coli* and other enteric bacteria are common if the intestine is the source of infection. Exploratory surgery may be required to diagnose a gastrointestinal rupture. The CBC results can be normal but often show an inflammatory leukogram and, in severe cases, a degenerative left shift.

Treatment

Treatment includes the prescription of appropriate antimicrobial agents, the administration of NSAIDs for pain and endotoxemia, and fluid support for dehydration. The prognosis is guarded, especially if an intestinal rupture has occurred.

Rectal Prolapse

Pathogenesis, Clinical Signs, and Diagnosis

Rectal prolapse is more common in sheep than in goats. This evagination of the rectal mucosa and rectal structures (and possibly the descending colon) usually is associated with excessive straining and short tail docking in lambs. Straining is seen in lambs with diarrhea caused by coccidiosis, *Salmonella* infection, or dietary imbalances; in ewes or ewe lambs with vaginal prolapse; in males with urolithiasis; and in animals grazing lush forage (particularly legumes such as alfalfa and clover). Rectal prolapse also can be secondary to chronic coughing, short tail docking, the use of growth implants, and rarely rabies.[5-11] In lambs, short tail docking (i.e., close to the body) appears to increase the incidence of rectal prolapse, as compared with long tail docking (at the level of the attachment of the caudal tail fold).[10,11] Thus short tail docking should be avoided. Regardless of the cause, after the rectal mucosa becomes everted and exposed, irritation of the mucosa causes further straining, which exacerbates the problem. Venous drainage of the prolapse may be compromised, but the arterial supply usually remains intact and contributes to the swelling.

Rectal prolapse is graded as type I to type IV, based on the extent of rectum and distal colon that is everted.[5] A description of these grades is presented in Table 5-3.

Treatment

Correction may be cost-prohibitive for feedlot lambs, and immediate slaughter is recommended. In more valuable animals, very mild, early cases can be treated with frequent application of hemorrhoidal ointment designed for humans and manual replacement of the prolapsed mucosa into the anus. In our own practice, we avoid applying pursestring sutures in the anus, because they tend to serve as a nidus for infection and result in further straining. If less aggressive therapies do not relieve the problem in 24 hours, however, placement of a pursestring suture may become necessary, particularly with type I and type II prolapse.

In all cases and with all modes of treatment, restricting feed for 24 to 48 hours while administering mineral oil is recommended. Dusty feedstuffs (concentrates, pellets, hay) should be avoided because they may contribute to coughing, which exacerbates this condition. Adding molasses to feeds and lightly wetting hay may help reduce problems with dust.

Placement of a pursestring suture is easily accomplished. The prolapsed tissue and perineal area are washed with mild soap and lubricated with petroleum jelly or hemorrhoidal ointment before the prolapsed mucosa is replaced.[5,9] After replacement of this tissue, the clinician inserts a tubular object (syringe case, wooden dowel, gloved finger) into the rectum. A pursestring suture of nonabsorbable suture material (3-5 nylon suture material) is then placed in the skin around the anus, tightened around the tubular object, and tied off. For placement of the suture, a cutting needle is used, entering and exiting at the 12 o'clock position. Tying the knot above the anus ensures that less fecal soiling of the suture will occur. The clinician should tie the suture in a bow knot to allow easy identification over the next few days and then remove the tubular object. The suture should be tight enough to prevent prolapse but loose enough to allow feces to pass. The clinician should regularly reevaluate the animal and if possible gradually loosen the pursestring suture at 24-hour intervals until no tension exists. After a full day of no tension, the suture can be removed. If the animal continues to strain, an epidural anesthetic can be administered. Petroleum jelly and hemorrhoid gel should be placed on the anus daily.[5,9]

The *injection of counterirritants* (1 mL or less of Lugol's iodine) around the rectum, either alone or in

TABLE 5-3 Grades of Rectal Prolapse

Grade	Description	Comments
Type I	Small, circular amount of submucosal swelling protrudes through anus; probing reveals a pocket or fornix just inside anus	Good prognosis in the absence of damage to mucosa *Repair*: Pursestring suture, iodine injection, submucosal resection
Type II	Slightly more circular submucosal and mucosal swelling, possibly containing retroperitoneal rectal tissue from anus; probing reveals a pocket just inside anus	Good prognosis with rapid treatment and no mucosal damage *Repair*: Pursestring suture, iodine injection, submucosal resection, rectal amputation
Type III	Complete prolapse containing part of the retroperitoneal structures of the rectum and the descending colon; probing reveals a fornix just inside anus; the affected portion of the descending colon does not prolapse through the anus	With vascular injury to the descending colon, prognosis is guarded to poor *Repair*: Submucosal resection and rectal amputation are the methods of choice
Type IV	The descending colon appears as a tube, and has intussuscepted through the rectum and anus; unlike the previous types, in this case a probe or finger can be inserted into the prolapse through the anal sphincter for a distance of 5 to 10 cm	With vascular injury to the descending colon, prognosis is poor *Repair*: Abdominal exploration may be required to determine the extent of damage to the descending colon

From Hooper RN: General surgical techniques for small ruminants: part II, *Presented at the Small Ruminants for the Mixed Animal Practitioner Western Veterinary Conference, Las Vegas, Nev, 1998.*

conjunction with anal pursestring suturing, is a quick and inexpensive treatment.[5,6,9] The clinician inserts an 18-gauge needle (5 cm) deeply into the skin around the anus at the 12, 3, and 9 o'clock positions. An injection at the 6 o'clock position should be avoided, because swelling around the urethra can result in obstruction.

For more severe cases, submucosal resection or amputation of involved rectal tissue may be necessary.[5,9] Rectal amputation can be performed using either a prolapse ring or a suture technique. Placement of a *prolapse ring* is a salvage procedure. The clinician inserts the prolapse ring into the rectum and places an elastrator band or suture around the area to be amputated, to induce vascular compromise and necrosis of tissue. If a ligature is used, it should be tightened to allow purchase on the tube or ring. A fibrosis is induced just proximal to the band or suture, and mucosa subsequently grows across the areas.[5] Strictures, peritonitis, and abscesses are possible complications, but this technique may be useful as a field procedure.

Submucosal resection can be performed with use of epidural analgesia after the prolapsed tissue and the perineal area have been surgically prepared. The clinician places two spinal needles (9 to 10 cm) at a 90-degree angle to each other 2 to 4 mm distal to the anal sphincter and through the entire depth of prolapsed tissue.[5]

A circular incision is made 2 to 4 mm distal to the spinal needles through the mucosa and around the outside of the anus. Another circular incision is made just distal to the caudal extent of the prolapse into the point at which the mucosa reflects on itself on the inner side of the prolapse. The clinician connects these two incisions with a longitudinal incision parallel to the prolapse and dissects the mucosa between the circumferential incisions.[5] The mucosal edges are then brought together using a suitable absorbable suture material in a simple interrupted pattern. After completion of this suturing, the clinician removes the two spinal needles and places a pursestring suture in the anal sphincter. Placement of the suture and follow-up care are the same as described for the pursestring suture technique. Submucosal resection decreases the incidence of both peritonitis and stricture formation compared with other surgical techniques, but it is costly to perform.[5]

With all of these techniques, use of a caudal epidural anesthetic (e.g., 2% lidocaine, 0.5 mL/45 kg of body weight) is recommended to decrease straining and ease pain associated with the procedure.[6,7] A xylazine epidural (0.01 to 0.03 mg/kg in a quantity sufficient to make 2 mL, with 2% lidocaine) may give longer relief (approximately 4 to 6 hours) from straining than that obtainable with lidocaine. An alcohol epidural also may prevent straining for extended periods. Either isopropyl alcohol or ethanol can be used to demyelinate the motor and sensory nerves.[5] This type of anesthesia can be permanent and therefore should be used only in animals intended for slaughter. Because of the potential for some loss of sciatic nerve function, the clinician should perform a test injection of a local anesthetic

(2% lidocaine) before using alcohol. If the epidural appears to be effective and no ataxia or muscle weakness of the rear limbs is noted, the clinician can inject a mixture of equal parts of lidocaine and alcohol into the sites where the test epidural was performed. Possible problems with alcohol epidural anesthesia include injection site necrosis, sciatic nerve dysfunction, and the inability to pass feces.[5]

Regardless of the type of epidural used, the clinician clips, washes, and dries the area before placing a small needle (20- to 21-gauge [2.6 cm]) in the cranialmost yet still movable intracaudal vertebral space—usually C1 to C2 or C2 to C3. The needle is placed on the dorsal midline, at 90 degrees to the skin, with the hub moved slightly caudally, and then slowly advanced (see Chapter 18).

Prevention

Management practices that predispose animals to rectal prolapse should be avoided. The most common association in lambs is with short tail docking.[10,11] The clinician should advise keepers and owners that docking closer than the attachment of the caudal tail folds should be avoided.[10,11] Other conditions to be avoided include dusty living or keeping quarters (pens, paddocks, or barns), overconditioned status (with excessively fat animals), and relevant disease processes—coccidiosis, internal parasites, respiratory disease, and urinary calculi. Although the estimated heritability for the incidence of rectal prolapse appears to be low (0.14),[11] the keeper should consider removing animals with a history of rectal prolapse, or whose offspring experienced prolapse, from the breeding flock or herd. Attention to good feeding practices and monitoring BCS also will aid in the prevention of rectal prolapse.

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DISEASES OF THE LIVER

Liver Abscess

Formation of liver abscesses usually is the result of chronic rumenitis in cattle, but these lesions are rare in sheep and goats. They may occur in feedlot lambs and kids and other animals fed rations high in grain. In lambs and kids, septicemia or extension of an umbilical vein infection can lead to formation of liver abscesses.[1] In most cases, however, liver abscess is an incidental finding. Weight loss, anorexia, depression, and decreased production (e.g., growth, milk) may be noted in affected animals.

In adults, *Corynebacterium pseudotuberculosis* is the most common pathogen. *Actinomyces pyogenes* and *Fusobacterium necrophorum* also are cultured from abscesses.[1,2] Liver enzymes may or may not be elevated. Diagnostic ultrasonography of the liver may help detect abscesses, especially if they are numerous and widespread. However, no specific treatment or control measure is available. Many of the preventive protocols used for feeder cattle can be applied to the control of abscesses in sheep and goats. Such strategies include slowly introducing concentrates into the diet, offering long-stemmed hay on a free-choice basis, and including rumen buffers (alkalizing agents) and antimicrobial agents in the feed.

Pregnancy Toxemia and Fatty Liver Syndrome

Pathogenesis

Fatty liver occurs in conjunction with pregnancy toxemia in ewes and does during the last month of gestation.[3,4] It is most common in both thin or obese ewes or does with a single large fetus, twins, or triplets.[1,3-5] During late gestation, particularly in obese females, the abdominal space is filled with accumulated fat and an ever-expanding uterus. Because of the lack of rumen space, these animals have difficulty consuming enough feedstuffs to satisfy energy requirements. In most management systems, late gestation occurs during the winter months, when less pasture is available and poorer-quality feedstuffs are offered. Energy requirements for ewes and does carrying twins or triplets are greatly increased during the final 2 months of gestation,

because 70% to 80% of fetal growth occurs during this time. Ewes with twins require 180% more energy, and those with triplets need 200% to 250% more dietary energy. Glucose maintenance in ewes pregnant with twins is significantly more prone to disturbance resulting in hypoglycemia than in ewes bearing singletons.[4] Pregnancy toxemia also occurs in association with anorexia caused by other diseases (e.g., foot rot, OPP, CAE) or sudden stresses (e.g., feed or weather changes, predator attacks, hauling). A period of anorexia or lack of sufficient energy intake will result in a negative energy balance. Affected animals begin to mobilize body stores of fat and transport them to the liver. In the liver, fat is catabolized to glycerol and free fatty acids (FFAs). FFAs can be used in the citric acid cycle (Krebs cycle) as an energy source, but not in the direct formation of glucose. Anorexic animals have less ruminal substrate available for production of the glucose precursor propionic acid. However, oxaloacetate, which is an integral part of the citric acid cycle, is removed from the cycle and converted into glucose. Depletion of oxaloacetate inhibits the normal citric acid cycle's function, thereby inhibiting the use of FFAs. As the pool of FFAs increases, they are converted to ketone bodies or repackaged into lipoproteins. Because ruminants are not efficient at transporting lipoproteins out of the liver and back to the adipose stores, the lipoproteins overwhelm the liver's ability to handle fats, leading to a massive buildup and resulting in a fatty liver. Because less substrate is available for glucose formation, more oxaloacetate is "cannibalized" from the citric acid cycle, further inhibiting the body's ability to use FFAs. This impairment in turn results in the continued accumulation of ketones. Hypoglycemia, hyperketonemia, and potentially uremia and death can occur.

Clinical Signs

Animals suffering from fatty liver or pregnancy toxemia become anorexic and depressed or dull, with altered behavior patterns, and may lag behind others in the group or become recumbent. Some are constipated, grind their teeth, have a ketone smell to the breath, demonstrate labored breathing or frequent urination, and suffer from dystocia. Neurologic signs include blindness, circling, incoordination, "star-gazing," tremors, and convulsions.[6-8] Death can occur if the condition is left untreated. In the case of fetal death in utero, maternal septicemia-endotoxemia and death are common sequelae.

Diagnosis

Diagnosis is based on clinical signs, the presence of multiple fetuses, and typical clinicopathologic findings.[3] CBC results may be normal or show an eosinophilia, neutropenia, and lymphocytosis. Affected animals may or may not be hypoglycemic, but ketoacidosis, hypocalcemia, and hypokalemia are common.[5-8] Liver enzymes usually are within normal limits but occasionally may be increased. Azotemia, both from dehydration and secondary to renal disease, is a common finding, and a fatal uremia may occur. Blood concentrations of β-hydroxybutyric acid greater than 7 mmol/L are consistent with pregnancy toxemia. Urinalysis will be positive for both ketones and protein.[3] Urine is collected from sheep by holding the nares and from does by frightening them and then allowing them a perceived escape, whereupon they stop, squat, and void.

Although not commonly performed, liver biopsy can help determine the extent of fatty infiltration. Serum protein pattern changes may become an available tool in the diagnosis of this condition in the future.[5] This syndrome must be differentiated from hypocalcemia, hypomagnesemia, polioencephalomalacia, encephalitis, lead toxicity, and cerebral abscesses.

Treatment

Very early cases (before onset of recumbency) may be treated with oral or intravenous glucose. A balanced electrolyte solution with extra calcium (25 mL of 23% calcium borogluconate/L), potassium (10 to 20 mEq/L), and 5% dextrose is needed.[3] In some cases, sodium bicarbonate is valuable in treating acidosis (see Chapter 3). Energy intake must be increased, and propylene glycol can be administered (15 to 30 mL every 12 hours) as a glucose precursor. Rumen transfaunation and supplementation with vitamin B complex (including vitamin B_{12}, biotin, niacin, and thiamine) also are recommended.

After affected females become recumbent, treatment must be very aggressive. Flunixin meglumine (2.5 mg/kg once daily) appears to improve survivability, but should be used in conjunction with other therapies.[3] Flunixin meglumine can be given daily in depressed anorexic animals, and its use appears to improve feed intake.[3] Researchers using recombinant bovine somatotropin showed a response, but it was not significant in comparison with that in control animals.[9] Removal of the fetuses is crucial in these cases. Chemically inducing parturition (by administering 2.5 to 10 mg of prostaglandin $F_2\alpha$ or 0.75 μg/45 kg of cloprostenol in does and 15 to 20 mg of dexamethasone in ewes) and giving the ewe or doe medical support (fluids, B vitamins, glucose) while waiting is a useful protocol in some cases. Unfortunately, during the time before parturition, endotoxemia from dead fetuses further compromises the female's well-being. For this reason, we recommend immediate cesarean section in depressed moribund animals (see Chapter 8). The owner should be forewarned of the poor prognosis for animals already in a moribund state. Fluid support during and after surgery is crucial.

Regardless of the therapeutic plan, the animal should be offered a palatable, energy-rich, highly digestible

feedstuff. The keeper and the clinician should take care to minimize the risk of a confounding disease during convalescence (e.g., lactic acidosis, polioencephalomalacia).

Prevention

Fatty liver and pregnancy toxemia can be prevented through proper management and nutrition. Maintaining animals in proper body condition throughout the year and making sure energy and protein levels are adequate in late gestation (see Chapter 2) are two key preventive measures.[3,6,7] The owner or manager should be taught to assess body condition in individual animals, avoid extremes in body condition, and maintain emergency stores of feed in case of severe weather or natural disasters. In overconditioned females, the keeper should be encouraged to restrict institution of weight loss programs to early gestation (if at all) and to avoid abrupt feeding changes, while promoting exercise (e.g., by increasing walking distances from mineral access to shelter). The requirement for energy may be one and a half to two times maintenance for dams with single fetuses and two to three times maintenance for those with multiple fetuses. Prevention of concurrent disease, which may further increase energy demands or cause anorexia (e.g., intestinal parasitism, foot rot), is crucial. The keeper should take care to increase the grain portion of the diet slowly, and ensure the consistent availability of fresh, clean water, as anorexia from rumen upset can lead to pregnancy toxemia. Ewes should be offered 0.5 to 1 kg of a cereal grain (corn, oats, barley, or a combination) every day during the final months of gestation; does can be offered ½ to 1 kg of grain. Keepers should maintain ewes and does at a body condition score of 2.5 to 3 (see Chapter 2) throughout gestation and evaluate the animals' energy intake every 2 to 4 weeks.

Ultrasonography can help identify females with multiple fetuses. These animals should be separated into groups and fed accordingly.[3] Ultrasonographic determination of fetal numbers is best accomplished between 35 and 90 days after breeding (see Chapter 8). Determination of fetal number may be enhanced with use of proper technique: shearing the hair or fiber in front of the udder, applying a coupling substance to the skin (e.g., alcohol, oil, lubbricating gel), and interrogating (viewing) as much of the abdomen as possible while systematically moving from one side of the posterior abdomen to the other, to obtain an appreciation of the abdominal structures including any fetuses present.

Animal keepers and clinicians should ensure that ewes are healthy and free of chronic diseases (e.g., OPP, CAE, foot rot, chronic parasitism) and that a good-quality trace mineral salt mixture is available on a free-choice basis. The addition of lasalocid (0.5 to 1 mg/kg/day) or monensin (1 mg/kg/day) to the feed or mineral mixture will enhance the formation of the glucose precursor propionic acid and improve the efficiency of feed use. Monensin should be used with caution, however, because associated toxicity has been reported; the agent should compose no more than 30 ppm of the complete diet. The inclusion of niacin (1 g/head/day) in a feed supplement or mineral mixture will help prevent pregnancy toxemia. Supplementation with lasalocid, monensin, or niacin should begin 2 to 4 weeks before the animals give birth.

Shearing in the last trimester also is recommended in ewes.[7] Many sheep producers routinely clip the wool around the vulva. If complete body shearing is performed, the incidence of fatty liver or pregnancy toxemia may be decreased, by several mechanisms: Sheared sheep require less energy to walk and graze. Sheared ewes also tend to shiver on cold days, exercising the enzyme systems that promote the more efficient use of FFAs as energy substrate. These ewes tend to seek shelter during cold weather, which may decrease lamb losses resulting from hypothermia. Obviously, if ewes are to be shorn, keepers should make adequate shelter available.

Keepers should avoid hauling or moving females during late gestation. Proper predator control measures should be maintained. Good hoof care programs should be in place on farms or ranches where grazing is the predominant form of nutrient intake. Sheep and goats should have their teeth checked to ensure good dentition before the breeding season. Animals with poor teeth should be culled.

Measuring serum β-hydroxybutyric acid concentrations is useful in assessing energy status in ewes. Values of 0.8 to 1.6 mmol/L suggest a negative energy balance. Keepers should take steps to correct the problem by feeding better-quality, more digestible feedstuffs.

White Liver Disease

White liver disease is a form of fatty liver disease reported only in Angora and Angora-cross goats and sheep. It is associated with cobalt deficiency.[10-14]

Pathogenesis

Cobalt is needed by rumen microflora to produce cyanocobalamin, or vitamin B_{12}, which is a coenzyme for methylmalonyl-coenzyme A (CoA) mutase. This enzyme is in turn needed to convert propionate to glucose through the Krebs cycle. Cobalt deficiency leads to the accumulation of methylmalonyl-CoA, or methylmalonic acid, which is converted to branched-chain fatty acids that accumulate in the liver. Diets high in grain, which is fermented to propionate, coupled with deficient or marginal cobalt intake, may predispose to this condition. White liver disease has not been reported in the United States, but ill thrift from cobalt deficiency has been observed. It is therefore possible that the disease goes unrecognized in some cases.[11-14]

Clinical Signs

Signs most commonly are seen in young animals and include ill thrift, anorexia, and diarrhea; sheep may exhibit photosensitivity. Clinical laboratory findings include a macrocytic-normochromic anemia and hypoproteinemia.[1,11,14]

Diagnosis

Abnormal serum or liver concentrations of vitamin B_{12} or liver cobalt levels are the basis for diagnosis. Liver cobalt concentrations of 0.08 ± 0.02 ppm determined on a dry matter basis were reported in goats with white liver disease, compared with 0.53 ± 0.11 ppm in control animals.[11,12]

Treatment and Prevention

Sheep can be treated with oral cobalt (1 mg/head/day) or vitamin B_{12} injections. The condition usually can be prevented by including cobalt in the ration by feeding a good-quality trace mineral salt. however in areas in which cobalt is extremely deficient or absent from all feedstuffs, the oral administration of cobalt-containing "bullets" along with supplementation with a cobalt-containing salt-mineral mixture, may be required.[13]

Copper Toxicosis

Pathogenesis

Copper toxicosis is more common in sheep than in goats.[1,6,8] Goats appear to excrete copper more efficiently than sheep and are more cow-like in their ability to resist toxicosis, but nevertheless are susceptible.[1,6,15-17] The use of copper oxide wire particles to treat internal parasitism has been suggested as a cause of copper toxicity in goats. Toxicity results from chronic accumulation in the liver from the ingestion of excess copper in relation to molybdenum or sulfate in the diet. In sheep, a copper-to-molybdenum ratio greater than 10:1 leads to the accumulation of excess copper. The most common sources of excess copper in sheep and goats are trace mineral mixtures and feeds formulated for cattle or horses. Clinical signs often are absent during the chronic accumulation phase. Onset of acute disease is related to the sudden release of copper from the liver in large amounts. Stress usually precipitates this acute phase. Acute release of copper and subsequent high blood copper concentrations cause an acute hemolytic crisis, resulting in anemia, hemoglobinuria, and acute renal failure. Existing hepatic disease (such as that caused by liver flukes) may predispose animals to this condition. Some breeds (e.g., Merino sheep) seem to be prone to copper absorption and storage problems, whereas others (e.g., pygmy goats) tend to be more resistant and prone to deficiency (see Chapter 2).

Clinical Signs

Anorexia, depression, diarrhea, and weakness all are signs of copper toxicity. In many instances, affected animals are found dead with hemolysis and icterus. Abdominal pain and diarrhea sometimes are present. Port wine–colored urine is evidence of hemoglobinuria. Hemoglobinemia produces icterus of the mucosal membranes and fever.

Diagnosis

Findings on clinicopathologic examination include anemia, hemoglobinemia, hyperbilirubinemia, increased liver enzymes, and azotemia. Urinalysis reveals hemoglobinuria and isosthenuria. The combination of azotemia and isosthenuria indicates acute renal failure. Definitive diagnosis of acute disease requires measurement of copper concentrations in serum. Normal blood copper concentrations are approximately 50 to 200 μg/dL in sheep and goats.[18] These concentrations increase 10- to 20-fold with an acute hemolytic crisis.[6] On necropsy, kidney copper concentrations are the most diagnostic tissue, because liver concentrations may be normal after release into the bloodstream. Generally, kidney concentrations greater than 100 ppm and liver concentrations greater than 350 ppm on a dry matter basis are diagnostic. If tissue copper is reported in wet weight, the conversion to dry tissue weight can be estimated by multiplying the tissue concentration by a factor of 3.5.

Treatment

Treatment of acutely affected animals often is futile. Appropriate management consists of supportive therapy for the acute renal failure and anemia and attempts to lower liver copper stores. Fluid therapy for the acute renal failure (see Chapter 3) is of clinical benefit, and a blood transfusion may be needed if the PCV drops precipitously. Ammonium tetrathiomolybdate (1.7 mg/kg IV or 3.4 mg/kg SC on alternate days for three treatments) is the most economical agent for treatment in acute cases. In valuable animals, oral D-penicillamine (26 to 50 mg/kg twice daily or 52 mg/kg once daily for 6 days) increases urinary copper excretion. Trientine is used in human beings but has shown variable results in sheep. Treatment of the remainder of the flock should include the oral administration of ammonium molybdate (50 to 500 mg/head/day) and sodium thiosulfate (300 to 1000 mg/head/day) for 3 weeks. Stress should be minimized, so keepers and clinicians should delay routine maintenance procedures such as deworming and hoof trimming until after treatment. When applicable, spraying a combination of ammonium molybdate and sodium sufate onto harvested forages low or deficient in copper to approximate the required therapeutic amount will decrease the stress required in daily oral dosing of chemicals. Allowing free access to grazing of forages high in sulfur (greater than 0.5% sulfur),

if available, for all surviving ambulatory animals also may help to minimize death losses in a flock or herd. Overzealous attempts to clear excessive hepatic copper stores may potentially lead to deficiency, excessively stress the animal, and can be costly, thus should be avoided. The offending source of copper should be eliminated. Caution should be taken in such cases to remove ionophores from the diet, because these agents may contribute to copper absorption. [19]

Prevention

Avoiding high dietary copper (more than 10 ppm), a high copper-to-molybdenum ratio (greater than 10:1) in the feed, use of copper-containing foot baths, and other sources of copper is crucial. Including supplemental molybdenum in the diet to lower the copper-to-molybdenum ratio to 6:1 to 8:1 is beneficial. Addition of up to 2 to 6 ppm of molybdenum may be required in many instances.

Often too much emphasis is placed on the trace mineral component of the diet. The clinician should be aware that even if no copper is added to the trace mineral mixture and the element does not appear on the product label, the mineral mixture may nevertheless contain copper. Many components of mineral mixes are contaminated with copper (zinc sulfate may contain 400 ppm of copper, dicalcium phosphate may contain more than 30 ppm of copper). Therefore the clinician needs to perform a dietary analysis to find and correct the problem.

Toxic Hepatitis

Pathogenesis

The liver is vulnerable to toxic insult because one of its major functions is detoxification. The most common plants that are gastrointestinal and liver toxins are shown in Table 5-4. Clinical signs will depend on the offending agent. Acute, severe toxicity is more common with chemical toxicosis, whereas plant toxins usually cause chronic disease. A thorough history is important, and in many cases, inspection of the animals' environment is required.

Clinical Signs

The clinical signs of toxic hepatitis can be subtle and nonspecific. Animals may exhibit only anorexia and depression. Icterus is more common with hemolytic diseases and is not always seen with liver disease. Photosensitivity is a common clinical feature in ruminants, and hepatoencephalopathy also can occur.

Diagnosis

Clinicopathologic data are more helpful in diagnosing acute toxicity. Serum AST and LDH levels can increase with hepatocellular necrosis, but such changes are not liver-specific, so muscle injury and disease must be ruled out. These enzymes also increase if serum is not separated from a blood clot in a timely fashion.[1] Increased levels of alkaline phosphatase (AP) and GGT indicate biliary stasis. AP concentrations also are not liver-specific, but increased serum levels of GGT are very specific for liver disease. GGT also increases in some hepatocellular diseases, so testing for normal concentrations is important.[18] Unfortunately, levels of all of these enzymes can be normal with liver disease, especially if it is chronic. Hyperbilirubinemia, hypoglycemia, low blood urea nitrogen (BUN), and hypoalbuminemia are not always evident, as is classically taught. If hepatoencephalopathy is suspected, blood ammonia concentrations may be elevated. Blood ammonia analysis may be impracticable in the field, because the blood should be kept on ice and the test should be performed within 30 minutes of collection. To enhance the accuracy of blood ammonia analysis, the clinician should collect blood from a normal control animal for comparison. Ammonia concentrations three times those in the control animal are diagnostic.[20] Liver biopsy remains the most valuable tool for diagnosing liver disease. Although clotting dysfunction may occur in liver disease, it is an uncommon complication in ruminants, and risk of bleeding should not discourage the clinician from performing a liver biopsy.

Treatment

If the intoxication is caught in the acute stage, activated charcoal (500 g in the adult animal) can be given. Supportive care, especially fluid support with dextrose solutions, is the mainstay of therapy. Low-protein diets may suppress ammonia production temporarily, but they can be detrimental over time, depending on the production status of the animal. Animals exhibiting photosensitivity should be housed indoors if possible, and broad-spectrum (systemic or topical) antibiotics may be necessary to control secondary bacterial dermatitis. Corticosteroids (e.g., dexamethasone 0.1 to 1 mg/kg IV or IM) may be indicated in early cases of photosensitization to decrease inflammation. Neurologic signs can be controlled with phenobarbital (initial dose: 10 to 20 mg/kg IV diluted in saline and administered over 30 minutes; subsequent doses: 1 to 9 mg/kg IV diluted in saline, as needed, up to three times daily). Diazepam (Valium) is contraindicated in hepatoencephalopathy because it may worsen deficits.[21]

Miscellaneous Liver Diseases

Congenital hyperbilirubinemia, or black liver disease, occurs in certain mutant Corriedale sheep.[1] The underlying disorder, the very similar to *Dubin-Johnson syndrome* in humans, is a genetically recessive condition characterized by an abnormality in the excretion

TABLE 5-4 Plants That May Cause Gastrointestinal or Hepatic Disease

Plant	Comments	Signs
Cocklebur	Erect annual herbage in sandy soils, flood plains, and overgrazed pastures; seeds are toxic	*Within hours to days of ingestion*: anorexia, vomiting, colic, dyspnea, gastroenteritis, chronic hepatitis, hepatic damage, death
Senecio (groundsel), *Crotalaria*, heliotrope, *Amsinckia* (fiddleneck), *Echium*	Pyrrolizidine alkaloids; excreted in milk and urine and can cross placenta; young more susceptible	Dullness, weakness, weight loss, icterus, fibrosis, hepatocytomegaly, bile duct proliferation, photosensitivity; subcutaneous edema, diarrhea
Lantana	Found in sandy, tropical areas; berries, leaves, and hay are toxic	*Chronic toxicity*—slow hepatic failure; icterus, photosensitization, weakness, bloody diarrhea, cholestasis, hepatic failure
Sneezeweed, bitterweed, rubberweed	Grows in overgrazed pastures; all parts of plant are toxic	*Acute toxicity*—gastrointestinal upset, depression, serous nasal discharge, salivation, bloat; *chronic toxicity*—vomiting, hepatic and renal congestion, gastric edema, aspiration pneumonia; pulmonary edema
Cabbage, kale, rape, mustard, wild mushroom	Remove from diet; add iodine to diet (for goiter)	Gastroenteritis, hepatic necrosis, photo-sensitization, goiter, hemolysis
Horsebrush	Stop grazing, keep animals indoors	Itching, uneasiness, inflamed eyes, blindness, serum discharge from scabs; degenerative changes in liver and elevated liver enzymes
Clover (crimson, red, subterranean burclover)		Photosensitization
St. John's wort	Perennial herb; grows along roadsides and in overgrazed fields; remove from diet and keep animals in shade	Increased respiration, diarrhea, pruritus, dermatitis, death
Blue-green algae	Toxic after a bloom	Vomiting, diarrhea, liver failure, photosensitization; necropsy findings include swollen bloody liver, edema around gallbladder, centrolumbar apoptosis, necrosis
Pokeweed		Vomiting, cramps, diarrhea, weakness, dyspnea, prostration, tremors, convulsions
Gossypol (cottonseed)	Toxicity seen in younger preruminants	Poor performance, convulsions, cardiac toxicity
Rhubarb	Contains oxalic acid	Gastrointestinal toxicity
Oak	Acorns and oak buds are most toxic	Abdominal pain, pseudomembranes in gastrointestinal tract, bloody diarrhea, depression, renal toxicity
Castor bean	Beans most toxic	Gastrointestinal irritation, bloody diarrhea, central nervous system disturbances
Mistletoe	Berries not toxic	Nausea, diarrhea
Other potentially pathogenic plants English ivy *Sesbania* Narcissus Elderberry Spurge Buckwheat Queen Anne's lace Milkweed Parsley, giant hogweed		

of conjugated bilirubin and phylloerythrin. Appearance of disease manifestations in animals often is related to consumption of green forage. Clinical signs include anorexia, photodermatitis, and icterus. Liver biopsy in affected animals reveals dark pink to black granules in otherwise normal hepatocytes. The syndrome first manifests itself in lambs around 5 months of age.[22]

A similar condition, termed *Gilbert's syndrome* in people, occurs in Southdown lambs around 6 months of age. It appears to be a recessive condition characterized by decreased hepatic uptake of phylloerythrin and bilirubin, with concurrent renal failure.[22] Clinical signs include icterus, photodermatitis, and ulceration around the ears and mouth. Liver biopsy reveals normal hepatic tissue. In both of these conditions, affected animals should be kept out of sunlight and fed minimal amounts of green forage. Obviously, these animals should be neutered or culled.

Various tumors of the liver, including fibrosarcoma, lymphosarcoma, and cholangiocellular carcinoma, have been reported in sheep and goats.[21,22] The use of ultrasonography and ultrasound-guided liver biopsy may aid in diagnosis.

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PATHOLOGIC CONDITIONS OF THE UMBILICUS

Umbilical Hernia

The umbilicus is an opening in the ventral abdominal wall that allows passage of the umbilical vessels and allantoic stalks. This opening should close within a few day of birth. The failure of this opening to close properly is termed *umbilical hernia.*

The hernial sac has an inner peritoneal layer and an outer layer of skin. Umbilical hernias probably are of genetic origin but may occur as sequelae to umbilical remnant infection. The opening in the abdominal wall is perceived as a ring on palpation. If the clinician can insert more than one finger into the hernial ring or if the hernia persists for more than 3 to 4 weeks, surgical intervention is indicated.

Pinning

Use of clamps or elastrator bands may be of value for closing small hernias (those less than 4 cm in diameter). The clinician should either lightly sedate the animal or infiltrate the skin around the hernia with a local anesthetic (e.g., 2% lidocaine). The animal is placed on its back and held by a technician helper. Any viscera prolapsing into the hernial sac should be replaced into the abdomen. The empty hernia sac and skin should then be tented away from the body wall to allow placement of an elastrator band as close as possible to the body wall. The clinician then inserts two metal pins (old fashioned baby diaper pins can be used) through the skin and hernia sac in a crossing fashion just distal to the elastrator band, so as to keep the elastrator band in place immediately adjacent to the external rectus sheath. Pinning in this fashion will result in ischemic

necrosis of the tissue distal to the elastrator band and enough inflammation of the tissue that the hernia ring adheres closed. The skin and hernia sac distal to the elastrator band will slough and the abdominal defect will heal in 7 to 14 days. Lambs should be given tetanus prophylaxis. This procedure and other clamping techniques are useful in females and some males. However, urine scalding of the skin may occur in some males. Clinicians should closely monitor animals that have undergone clamping for signs of abdominal discomfort or wound complications.

Surgical Resection

In cases in which the hernial ring is larger than 5 cm, surgical intervention will yield the most reliable results. The animal can be sedated and the skin of the umbilical region then infiltrated with a local anesthetic, or general anesthesia can be used. The area around the hernia is clipped and surgically prepared. The clinician makes an elliptical skin incision around the hernia sac and dissects down to the hernia ring at the external rectus sheath. The abdominal cavity is opened just cranial (or caudal) to the hernia ring on the linea alba to allow introduction of a finger into the abdominal cavity. The clinician uses this finger to digitally palpate the hernia ring to ensure that no viscera have adhered to the inner lining of the ring and that no enlarged or infected umbilical remnants (umbilical vein, umbilical arteries, or urachus) are present. The surgeon then carefully excises the hernial sac at the hernial ring. Any adhesions or abnormal umbilical remnants present are then excised before closure of the defect in the abdominal wall. This closure can be accomplished by simply opposing the incised edges of the external rectus sheath with absorbable suture in a simple continuous pattern. In the case of large hernias with tension on the body wall closure, a near-far-far-near suture pattern may be used. As a matter of personal preference (that of A.N.B.), a near-far-far-near suture pattern can be used in the middle of the incision to relieve tension, with completion of the closure in a simple continuous suture pattern. This approach provides a secure repair with rapid healing. The subcutaneous tissue should be closed with absorbable suture in a simple continuous pattern, and the skin can be closed using whatever pattern the clinician prefers. Animals should be given tetanus prophylaxis and antibiotics. They should be closely monitored for signs of sepsis and surgical failure. Exercise should be limited for 7 to 14 days after surgery.

Umbilical Infections

Infections of the umbilical arteries (omphaloarteritis) and veins (omphalophlebitis) and urachal disease can occur as a consequence of failure or partial failure of passive transfer of colostral antibodies and subsequent sepsis. Contamination of the umbilicus, retracting of these structures after stretching and breaking, and chemical damage (from strong tincture of iodine) to the amniotic remnants are other possible causes.[1-3] Dipping the umbilicus in iodine or iodine-chloriodine solutions is a common practice. Aggressive use of these chemicals, however, may precipitate severe inflammation of the cord. Excessive torsion of the umbilical cord, distention of the proximal urachus, and some genetic factors all may be associated with patent urachus, which also may occur as a sequela to omphaloarteritis or omphalophlebitis.

Clinical Signs and Diagnosis

The clinical signs include umbilical swelling, pain, and occasionally drainage or discharge of the umbilical stump. Palpation and transabdominal ultrasonographic evaluation will reveal an enlarged cord-like structure ascending from the umbilicus cranially (the umbilical vein) or caudally (the urachus or umbilical artery). Ultrasonographic evaluation may indicate presence of an abscess or thickened tissue. Patent urachus is associated with dermatitis, urine scalding of the ventral abdomen, and urine dribbling. If the urachus becomes infected, it may leak urine intraperitoneally or subcutaneously. Both of these developments may be identified by abdominal palpation, ballottement, ultrasonographic evaluation, and, when indicated, paracentesis.[1] The CBC may reveal neutrophilia. Blood culture is indicated if sepsis occurs simultaneously. Occasionally, infection of the internal structures may occur with no outward umbilical swelling. Deep abdominal palpation and the use of real-time ultrasound imaging are necessary to obtain a diagnosis. Animals with umbilical infections also may exhibit signs of septicemia, anorexia, depression, joint distention, and fever.

Treatment

Repair of Patent Urachus. If a patent urachus occurs without inflammation of the associated tissues, it can be cauterized daily with iodine or silver nitrate. However, if it remains patent for more than 5 days, it should be surgically closed. For the surgical repair procedure, the animal should be placed under general anesthesia (see Chapter 18). The area around the umbilicus is clipped and surgically prepared, and broad-spectrum antimicrobial therapy is instituted 2 to 4 hours before surgery. The clinician opens the abdomen lateral to the umbilicus and digitally explores the adjacent area for adhesion formation. The urachus should be identified and followed to the urinary bladder. The urachal attachment to the bladder is then amputated, and the bladder is closed using a double-layered inverting (Cushing) pattern. The abdominal wall, subcutaneous tissue, and skin are closed as described for umbilical hernia repair.

Medical versus Surgical Management of Infection. On occasion, some cases of omphalophlebitis-omphaloarteritis can be effectively treated medically. Prolonged antibiotic therapy with a broad-spectrum antimicrobial agent (ceftiofur, 2.2 mg/kg once a day, or oxytetracycline, 20 mg/kg SC every 72 hours) may be attempted. If medical therapy is ineffective, however, the infected umbilical remnants should be marsupialized or excised. We prefer more aggressive surgical removal of the umbilical remnants. As with urachal surgery, the abdomen should be opened lateral to the umbilicus. Depending on the severity of infection and the amount of tissue involved, the clinician may need to perform extensive dissection of necrotic tissue and possibly intestinal resection.[3]

Surgical Management of Extensive Infection. If the infection of the umbilical vein extends to and involves the liver, marsupialization of the umbilical vein is an effective method of therapy.[2,3] The clinician can pull the vein to the cranialmost portion of the abdominal incision and suture it to the muscle layers and skin before closing the abdomen as described for umbilical hernia repair. Our own preference, however, is to cover the transected end of the umbilical vein (with a sterile glove or surgical sponge) and pull it through a stab incision cranial to the hernia. It can then be secured in place with suture. The abdominal incision is then closed as described in hernia repair. This method of repair will minimize the incidence of abdominal wall herniation. Only monofilament, absorbable, non–gut suture material should be used.[3] The venous stump should be flushed daily with antiseptic solution (1% chlorhexidine or 0.1% povidone-iodine), and the animal should be maintained on antibiotics for more than 14 days. The venous stump usually closes within a month.[3] Very rarely, a second operation may be required to resect the previously infected umbilical vein.

Prevention

Umbilical infections can be prevented or their incidence drastically reduced by ensuring adequate intake of good-quality colostrum. In addition, lambs and kids should be exposed to only minimal stress (particularly during the first 2 to 3 days of life), to enhance colostral absorption. In some management scenarios, dipping of the navel with noncaustic materials also helps reduce the incidence of such infections.

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