GI CONSULT: What the Specialists Share When Patients Can’t Keep Food Down
CANINE CODE BLUE: THE 9-1-1 NATURE OF GDV AND SAGO PALM POISONING

Gastric Dilatation–Volvulus Syndrome

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Summary
Critical Keys
Etiology
Emergency Treatment
Imaging Studies
Surgery
*Incisional Gastropexy*
*Belt Loop Gastropexy*
Prognosis
Postoperative Management
Chronic Intermittent Gastric Volvulus
References and Suggested Reading
VIDEO – Surgical and Gastric Repositioning, Incisional Gastropexy, and Belt Loop Gastropexy in GDV Cases
CONTENTS continued

Sago Palm Toxicosis
Todd R. Tams, DVM, DACVIM • Justine A. Lee, DVM, DACVECC, DABT

Summary
Critical Keys
Plant Identification
Mechanism of Toxicity
Clinical Signs
Clinicopathologic Testing
Treatment
Prognosis
Conclusion
References

FEEDBACK ON FOOD BACK: MEGAESOPHAGUS IN DOGS, ESOPHAGEAL STRicture IN CATS

Megaesophagus with Myasthenia Gravis
Todd R. Tams, DVM, DACVIM • David S. Biller, DVM, DACVR • Howard B. Seim III, DVM, DACVS

Summary
Critical Keys
Megaesophagus and Myasthenia Gravis
Clinical Signs Associated with Megaesophagus
Diagnosis
Diagnostic Imaging
  Contrast Study: Esophagram
  Radiographic Changes with Megaesophagus
  Esophagram/Barium Swallow Changes with Megaesophagus
Laboratory Tests
Treatment
General Management Principles for Megaesophagus
A Recent Advance: Use of Special Feeding Chairs
Gastric Feeding Tube Placement
Megaesophagus Client Information and Support Group
Feline Esophageal Stricture
David C. Twedt, DVM, DACVIM • David S. Biller, DVM, DACVR • Howard B. Seim III, DVM, DACVS
Summary
Critical Keys
Imaging
Survey Radiographs
Contrast Studies
Endoscopy
Treatment
Balloon Dilation
References and Suggested Reading

CRISIS VERSUS CHRONIC: HOW THE CAUSE AFFECTS THE CASE OF THE VOMITING DOG
Acute Pancreatitis in Dogs – To Feed or Not to Feed
Todd R. Tams, DVM, DACVIM • David S. Biller, DVM, DACVR • Howard B. Seim III, DVM, DACVS
Summary
Critical Keys
Etiology
Risk Factors
Diagnosis
Laboratory Findings
Radiology
Ultrasonography
Ultrasonography Findings in Pancreatitis
Cytology
CONTENTS continued

Histopathology
Medical Management
Why Is Early Feeding Important?
Feeding Dogs with Pancreatitis
Nasoesophageal Tube Placement
  - Esophagostomy Tube Placement
  - Eld Esophagostomy Tube Placement Technique
  - Curved Carmalt Hemostat Technique
Other Potential Therapies
Surgical Management of Pancreatitis
References
VIDEO – Eld Esophagostomy Tube Placement

**Gastric Antral Mucosal Hypertrophy**

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Summary
Critical Keys
Imaging Studies
Normal
Contrast Studies
Ultrasound
Surgery
Pyloromyotomy
Transverse Pyloroplasty
Y-U Pyloroplasty
Pylorectomy
Prognosis
References and Suggested Reading
VIDEO - Pylorectomy
SKINNY, SKINNY KITTY: IS IT IBD OR NEOPLASIA?

Inflammatory Bowel Disease and Intestinal Lymphoma in Cats

Todd R. Tams, DVM, DACVIM • David S. Biller, DVM, DACVR • Howard B. Seim III, DVM, DACVS

Summary
Critical Keys
Abdominal Imaging – IBD Versus Lymphoma

Radiology
- Small Intestinal IBD
- Intestinal Lymphoma

Ultrasonography of the Small Intestines
- Intestinal Ultrasound: IBV Versus Lymphoma
- Ultrasound of IBD
- Ultrasound of Intestinal Lymphoma
- Ultrasonographic Evaluation of Muscularis Propria in Cats with Diffuse Small Intestinal Lymphoma or IBD

Intestinal Biopsy Techniques

Endoscopic Biopsy

Surgical Biopsy Techniques for Abdominal Organs
- Intestinal Biopsy
- Lymph Node biopsy
- Liver Biopsy

Treatment of IBD
Pharmacotherapy
Cobalamin Therapy in Cats

What If Biopsies Are Not Definitive for Either IBD or Small Cell Lymphoma?

Treatment of Intestinal Lymphoma in Cats

References

Table 1. Ultrasonographic Measurement of Feline Abdominal Lymph Nodes
Table 2. Pharmacotherapy of IBD in Cats
VIDEOS – Intestinal Biopsy, Liver Biopsy, Pancreatic Biopsy
CONTENTS continued

Feline Intestinal Adenocarcinoma 61
David C. Twedt, DVM, DACVIM • David S. Biller, DVM, DACVR • Howard B. Seim III, DVM, DACVS

Summary
Critical Keys
Imaging Studies
Radiology
Ultrasonography
Adenocarcinoma
Surgery
Postoperative Care
Prognosis
Suggested Reading
VIDEO – Intestinal Anastomosis in Cat with Intestinal Adenocarcinoma
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Gastric Dilatation-Volvulus Syndrome

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SUMMARY

Gastric dilatation-volvulus syndrome (GDV) is an acute life-threatening condition characterized by malposition of the stomach with rapid accumulation of swallowed air in the stomach and the inability to expel the gas. The increased gastric pressure can result in hypovolemic shock. Without rapid decompression and treatment, the mortality rate can be as high as 15% to 33% and is often a cause for “acute death” in dogs. Current emergency management strategies, prevention, complications, surgical techniques, and prognosis of GDV are covered in this article.

CRITICAL KEYS

• Emergency therapy is directed at decompression of the stomach and management of hypovolemic shock. Initial fluid resuscitation with crystalloids is imperative to improve cardiovascular stabilization and overall perfusion.

• The key to success is gastric decompression via either orogastric intubation or trocharization. Trocharization into a tympanic area of the stomach (using sterile technique) is less invasive and equally as effective.

• Simple blood tests (e.g., minimum database) and point-of-care testing (e.g., lactate) can help guide therapy and may help evaluate GDV complications and/or predict survival.

• Negative prognostic factors include gastric viability, disseminated intravascular coagulation, cardiac arrhythmias, and peritonitis.

• Only after the patient is stabilized are radiographs warranted to aid in diagnosis. Imaging of the stomach is indicated if a volvulus is in question or if foreign body ingestion or other intra-abdominal disorder is suspected.

• Chest radiographs should also be considered to rule out underlying pathology (e.g., aspiration pneumonia, cardiac disease, metastasis), based on the stability of the patient.

• Surgical exploratory for patients suspected of having a GDV is considered an emergency procedure. The longer the stomach is malpositioned, the more at risk the stomach is for developing ischemia and possible necrosis. Surgical intervention involves repositioning of the stomach and performing a permanent gastropexy.

• In the immediate postoperative period, in addition to standard fluid and electrolyte management, additional therapy involving analgesics, antiemetics, acid blockers, and gastric prokinetic agents may be indicated.

• Nutritional management includes feeding multiple small meals a day of a highly digestible low fiber diet.

• A basic thumb rule is if the patient can walk into the hospital on its own the prognosis is much better compared with the recumbent semi-comatose dog, which has a guarded to poor prognosis.

• Recent studies show that the use of lidocaine in GDV patients may improve survival.
ETIOLOGY
The etiology of gastric dilatation-volvulus (GDV) syndrome is unknown; however, a fundamental abnormality involves laxity of hepatoduodenal and hepatogastric ligaments leading to a high degree of mobility of the stomach. Some suggest that an underlying gastric hypomotility in large deep-chested dogs (e.g., Great Dane, Standard Poodle, German shepherd) results in chronic gastric distention, stretching of supporting ligaments, and then secondary volvulus of the stomach. In high-risk breeds undergoing elective procedures (e.g., ovariohysterectomy), a prophylactic gastropexy is typically recommended to prevent volvulus; however, keep in mind that this does not always resolve gastric distention.

EMERGENCY TREATMENT
Emergency therapy is directed at decompression of the stomach and management of resultant hypovolemic shock (e.g., tachycardia, hypotension, weakness, collapse, pallor); this is due to compression of the abdominal caudal vena cava with lack of venous return to the heart and decreased cardiac output. The substantial decrease in cardiac output results in poor perfusion and oxygen delivery to many tissues. Cardiac arrhythmias (e.g., ventricular premature contractions [VPCs] or idioventricular rhythm) are also common, likely due to myocardial ischemia or secondary splenic involvement. With GDV, clinical signs of hypovolemic shock may be worsened secondary to hemorrhagic shock; due to the gastrospenic attachments, avulsion of the short gastric vessels may result in hemorrhage and secondary hemoabdomen. Likewise, splenic torsion or infarction of splenic arteries and thrombosis of splenic veins results in splenic necrosis. Untreated, these secondary complications may further contribute to poor perfusion and cellular hypoxia, predisposing the patient potentially to systemic inflammatory response syndrome (SIRS), disseminated intravascular coagulation (DIC), and less commonly, multiorgan dysfunction (MODS). Additional possible contributing factors include pooling of blood (e.g., in the caudal vena cava, portal vein, splanchic circulation), tissue hypoxia, metabolic acidosis, endotoxemia, and sepsis.

The stomach is the organ most severely affected by ischemia and decreased gastric perfusion, resulting in serosal hemorrhage and edema of stomach wall. This typically is seen near the area of the fundus and may spread to the body. Likewise, ischemia may involve the esophagus, resulting in a poorer prognosis. Severe compromise to the gastric wall can result in necrosis and perforation, with resultant pneumoperitoneum and secondary septic peritonitis. According to dePapp et al, a plasma lactate concentration > 6.6 mmol/L has been associated with gastric necrosis and a worse outcome. More importantly, serial lactate parameters should be evaluated, as persistently elevated lactate levels are more likely to be significant than interpretation of a single lactate level measurement.

The most important goal of treatment is correction of circulatory collapse with intravenous (IV) fluid therapy with a balanced, buffered maintenance crystalloid. While most of these patients require aggressive IV fluid therapy, the use of the whole shock dose (“60–90 mL/kg”) of fluids is typically not required. Most patients respond well to the use of smaller aliquots of a crystalloid (e.g., 1/3 of a shock bolus, followed by repeat boluses as needed), followed by a colloid if necessary (e.g., 5 mL/kg Hetastarch). Decompression of the stomach—either in the form of trocharization of the stomach or orogastric intubation—can be performed. Both are effective, although trocharization into a tympanic area of the stomach (using sterile technique) is less invasive. Only after correcting the circulatory collapse and decompression of the stomach is surgery indicated for repositioning and pexying the stomach.

Other ancillary treatment includes the use of anti-arrhythmics (e.g., lidocaine, procaainamide), antiemetics (e.g., maropitant, ondansetron), nutritional support, prokinetics, and analgesics (please see Postoperative Care for more information). Recent studies show that the use of lidocaine in GDV patients may improve survival. Prophylactic antibiotics are also somewhat controversial, but rational arguments are made for their use.

Only after the patient is stabilized are radiographs warranted to aid in diagnosis. Imaging of the stomach is indicated if a volvulus is in question or if foreign body ingestion or other intra-abdominal disorder is suspected.

IMAGING STUDIES
Radiography can help to differentiate between gastric dilation and GDV. If radiography is necessary to determine the diagnosis, do not perform it until the patient is stable. Gastric dilation is defined as a moderately to severely distended stomach filled primarily with gas, gas and fluid, fluid, or ingesta. Most importantly, the enlarged stomach retains its normal position.

Gastric volvulus is differentiated from gastric dilation by displacement
(abnormal position) of the stomach due to its rotation. The stomach may be distended; these animals may present in acute crisis, tympanic, and retching. The location of the pylorus and duodenum dorsocranial and near or to the left midline is the most common appearance to this malposition. If questionable, a low-volume positive contrast gastrogram and a left lateral abdominal film can be performed to evaluate positioning of the pylorus. A standard right lateral radiograph (view of choice) should fill the pylorus with gas in an animal with GDV. The dorsoventral radiographic view is also preferred in order to facilitate filling the abnormally displaced pylorus with air so that it can be easily identified. On a right lateral view of a dog with GDV, the pylorus lies cranial to the body of the stomach. On the dorsoventral view, the pylorus appears as a gas-filled structure to the left of midline.

Other radiographic changes that can be seen include compartmentalization (radiographic recognition of soft tissue bands that project into or across the gas-filled lumen of the rotated stomach). The appearance of the stomach has been described as a “double bubble,” boxing glove sign,” or reverse “C.” These soft tissue bands are due to folding of the stomach on itself as the folded wall projects into the lumen and is outlined by gas within the lumen. Splenomegaly and variations in the location of the body of the spleen due to torsion may also be seen. The gastric wall may be thin. Gas may be seen within the gastric wall or liver (portal vessels) due to necrosis of the wall. Reduced size of the caudal vena cava and cardiac silhouette (due to reduction in preload), esophageal dilation (megaesophagus), and reflux paralytic ileus in the small intestine (which may be due to pain as well) may be present. With necrosis/rupture of the gastric wall, loss of abdominal detail (peritonitis) and free gas may be observed.

SURGERY*

A video demonstrating surgical and gastric repositioning, incisional gastropexy, and belt loop gastropexy in GDV cases can be viewed at http://www.gloydgroup.com/proceedings/videos/2014-gi-symposium-GDV-canine. The video is provided courtesy of Dr. Howard B. Seim III.

Surgical exploratory and gastric repositioning is considered an emergency procedure. Delaying surgery may further encourage gastric ischemia and subsequent necrosis. If the surgeon does not have an assistant, it is vital that the abdomen is opened from xyphoid to pubis and a self-retaining abdominal retractor be used to maintain exposure. Exteriorizing the enlarged spleen and other viscera may be necessary to facilitate repositioning the stomach.

Anatomic repositioning of the stomach is necessary to perform prior to permanent gastropexy. Repositioning occasionally occurs spontaneously at the time of gastric decompression. Knowledge of normal anatomy is necessary to understand how repositioning is performed. The pylorus, located near the cardia of the stomach, is grasped by one hand and elevated as the other hand presses down on the fundus and body of the stomach. Reduction is generally easily performed if the stomach has been adequately decompressed. The spleen is generally returned to its normal location once the stomach has been successfully de-rotated. Splenectomy is rarely performed but may be necessary if splenic vessels are infarcted. The greater curvature and fundus of the stomach should be examined for areas of necrosis. If necrosis is present, partial gastrectomy may be necessary.

Incisional Gastropexy

This technique is based on the construction of a seromuscular antral flap attached to a scarified segment of transversus abdominus muscle. A 2- to 3-cm incision is made in the antral portion of the stomach. The bleeding surface of the antrum is brought to the right body wall. With the stomach in a normal position, the bleeding antral surface is touched to the peritoneal wall to create a blood mark on the peritoneum. This is the location of the gastropexy. The peritoneum and transverses abdominus muscle are incised creating a mirror image defect of the stomach flap. The stomach flap incisional defect is sutured to the abdominal wall incisional defect with two rows of simple continuous or simple interrupted synthetic absorbable suture. Advantages of the incisional gastropexy include the following: it is easy to perform if you do not have an assistant in the operating room, it can be performed quickly, and it is an effective means of permanent gastropexy. This technique is the one recommended by the authors.

Belt Loop Gastropexy

This technique is based on the construction of a seromuscular antral flap attached around a segment of transversus abdominus muscle. A horseshoe-shaped incision is made in the serosal layer of the antral portion of the stomach with its base at the greater curvature. The seromuscular
portion of the stomach is identified by grasping full-thickness antral wall between the thumb and index finger and “slipping” the mucosal and submucosal layers away so only the seromuscular portion of the wall remains between thumb and finger. The seromuscular layer is incised with scissors and the horseshoe-shaped seromuscular antral flap is dissected and elevated of the submucosal layer. The stomach is replaced in the abdominal cavity in normal position and the seromuscular flap lined up with the transversus abdominus muscle. Once this optimal location is discovered, two longitudinal incisions (along the fibers of the transversus muscle) are made in the transversus abdominus muscle. The segment of muscle between the incisions is undermined. The seromuscular flap from the stomach (i.e., belt) is passed through the transversus abdominus m. (i.e., loop) and sutured to itself to complete the “belt loop” gastropexy. Use 2-0 or 3-0 monofilament absorbable synthetic suture in a simple interrupted or continuous pattern to secure the flap in place. Advantages of belt loop gastropexy include the following: it is relatively easy to perform alone and in the middle of the night, it can be performed quickly, and it is an effective means of permanent gastropexy.

**PROGNOSIS**

Patients that are admitted to the hospital early, successfully decompressed, treated for shock, and operated urgently have a favorable prognosis for full recovery. Patients in which presentation is delayed, however, are more likely to develop serious ischemia-reperfusion injury, cardiac arrhythmias, and ongoing gastric ischemia and necrosis and thus have a guarded to poor prognosis. A basic thumb rule is if the patient can walk into the hospital on its own, the prognosis is much better compared with a recumbent semi-comatose dog, which has a guarded to poor prognosis.

**POSTOPERATIVE MANAGEMENT**

In the immediate postoperative period, in addition to standard fluid and electrolyte management, additional therapy involving analgesics (e.g., opioids), antiemetics (e.g., maropitant, ondansetron), acid blockers (e.g., famotidine), and gastric prokinetic agents (e.g., metoclopramide, cisapride) may be indicated. Cisapride is recommended as the prokinetic of choice. Nutritional management includes feeding multiple small meals a day of a highly digestible low fiber diet. Some recommend liquefying the meal to improve gastric emptying and chronic prokinetic therapy may be required in some cases on a long-term basis as well.

**CHRONIC INTERMITTENT GASTRIC VOLVULUS**

We will occasionally see a large deep-chested breed dog with intermittent vomiting, abdominal discomfort, and or abdominal distention. This syndrome is associated with intermittent malpositioning of the stomach but without complete volvulus. Routine abdominal radiographs may show a stomach in normal position but subsequent radiographs with or without contrast in various positions may demonstrate abnormal position or movement of the antrum (a “floppy” stomach). In these cases, a prophylactic gastropexy is advised in addition to dietary and or prokinetic agents dictated by clinical response.

**REFERENCES AND SUGGESTED READING**


VIDEO – Surgical and Gastric Repositioning, Incisional Gastropexy, and Belt Loop Gastropexy in GDV Cases
Sago Palm Toxicosis in Dogs
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SUMMARY

While veterinarians are aware of the dangers of lilies to cats, many are unaware of the dangers of sago palm to dogs. This tropical plant is commonly found in the southwestern and southeastern United States. The prevalence of sago palm toxicosis in dogs has increased, likely due to the increased availability of the bonsai houseplant variety that is now sold throughout North America. Ingestion results in acute gastrointestinal (GI) signs (e.g., vomiting, diarrhea, hypersalivation) within 15 minutes to several hours after ingestion, with severe acute hepatic necrosis and neurologic signs (e.g., weakness, ataxia, seizures, tremors) within 2 to 3 days after ingestion. The latest information on clinical presentation, management, and treatment of the acute hepatic necrosis associated with sago palm toxicosis is presented in this article.

CRITICAL KEYS

- There are many causes of acute vomiting in puppies, including infection (e.g., viral, bacterial, parasitic), dietary indiscretion, foreign body ingestion, and toxic ingestion. Appropriate history is imperative to help identify the underlying cause.
- While the majority of plants ingested only result in gastrointestinal signs (e.g., vomiting, diarrhea), a few can be life threatening. Veterinary professionals must be aware of the dangers of sago palm toxicosis.
- Sago palm typically thrives in hot, humid climates and is commonly found in the southern United States and Hawaii. However, sago palm is now more commonly sold as an indoor bonsai houseplant in locations all around the country, and exposure to pets is now more common.
- Sago palm toxicosis affects three main organ systems: the gastrointestinal tract (GIT), the liver, and the central nervous system (CNS). Acute hepatic necrosis (AHN) can be severe, with a reported mortality of 50% in recent veterinary literature.4
- Aggressive decontamination, including emesis induction and administration of multiple doses of activated charcoal, is recommended due to the severity of toxicosis.
- The ancillary use of benign hepatoprotectants (e.g., N-acetylcysteine, SAMe, milk thistle) is recommended due to the severity of hepatic injury.
- Studies showed that survivors of sago palm toxicosis were more likely to have received activated charcoal, which reiterates the importance of aggressive decontamination with this toxicant.
- Overall, long-term recovery is poor, as the potential for chronic liver disease exists.
While there are thousands of species of plants, very few result in fatalities when ingested by dogs. Veterinarians must be aware, however, of the few plants that can result in severe morbidity and mortality. In veterinary medicine, sago palm is one of the most deadly plant ingestions seen, as it can result in severe AHN.

In any patient presenting with AHN, rule outs include:

**Toxicological causes:**
- Acetaminophen/paracetamol
- Nonsteroidal anti-inflammatory drugs (NSAIDs; typically chronic or subacute exposure)
- Xylitol
- *Amanita* mushrooms
- Blue-green algae
- Sago palm

**Underlying diseases:**
- Chronic hepatopathy
- Cholangiohepatitis
- Cirrhosis
- Extrahepatic biliary duct obstruction (e.g., pancreatitis)
- Anatomic abnormalities (e.g., portosystemic shunts, microvascular dysplasia)
- Neoplasia

**PLANT IDENTIFICATION**

Sago palms are technically not “palms” and belong to a group of plants dating back to the dinosaur era. Sago palms are often referred to as “living fossils” and are naturally found in tropical/subtropical environments. In the United States, sago palm grows in the southern states and Hawaii; however, it is also grown as an ornamental bonsai houseplant and thus can be found anywhere. Veterinarians should be aware that the plants are sold at many garden centers including Lowe’s and Home Depot.

These plants are members of the Order Cycadaceae, genera Cycads, Macrozamia, and Zamias. Examples of the cycad family include Cycad (*Cycas cirinalis*), Japanese cycad (*Cycad revoluta*), coontie plant (*Zamia pumila*), and cardboard palm (*Zamia furfuracea*). All parts of sago palm are considered poisonous, with the seeds (nuts found on the female plant) being the most toxic part of the plant, as they contain large amounts of the toxicant, cycasin. As little as two seeds can cause clinical signs in dogs.

**MECHANISM OF TOXICITY**

While the exact mechanism of toxicity of sago palm is not entirely clear, the toxicokinetics are thought to be due to three primary toxins. The primary active toxic agent is an azoglycoside called cycasin, which results in hepatotoxicity. In the GIT, cycasin is converted to methylazoxymethanol (MAM) by a β-glucosidase. The second toxic agent is β-methylamino-L-alanine (BMAA), which is considered a neurotoxic amino acid. Finally, the third toxic agent has not been identified, but is known to have a high molecular weight.

**CLINICAL SIGNS**

Sago palm toxicosis targets three main organ systems: the GIT, liver, and CNS. Ingestion results in acute GI signs (e.g., vomiting, diarrhea, hypersalivation) within 15 minutes to several hours after ingestion. Severe acute hepatic necrosis and CNS signs (e.g., weakness, ataxia, seizures, tremors) can be seen within 2 to 3 days after ingestion.

Clinical signs or physical examination findings include:
- Anorexia
- Vomiting (e.g., hematemesis)
- Diarrhea (e.g., melena, hematochezia)
- Generalized malaise
- Dehydration
- Ascites
- Abdominal pain
- Icterus
- Melena
- Ataxia
- Tremors
• Seizures
• Coma
• Death

**CLINICOPATHOLOGIC TESTING**

In patients exposed to sago palm, routine blood work (e.g., complete blood count, chemistry panel, urinalysis) should be performed on presentation. Baseline blood work [e.g., packed cell volume/total solids (PCV/TS), blood glucose, chemistry] should be performed daily while the patient is hospitalized. In patients with evidence of hepatic injury, coagulation parameters [e.g., prothrombin time (PT), activated partial thromboplastin time (aPTT), platelet count], ammonia, and bile acids should also be obtained.

Clinicopathologic findings in dogs with AHN secondary to sago palm toxicosis typically reveal elevations in liver enzymes [e.g., aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALK PHOS)] and bile acids; however, elevations may not occur for 24 to 48 hours, and may take up to 2 to 9 days to be seen. Other findings include hemoconcentration or anemia, hypoalbuminemia, hypocholesterolemia, hyperbilirubinemia, hyperglycemia or hypoglycemia, hyperlactatemia, leukocytosis, thrombocytopenia, and coagulopathy (e.g., prolonged PT, aPTT). Lastly, increased blood urea nitrogen (BUN, due to pre-renal azotemia) or decreased BUN (secondary to liver failure) may be seen.

Histopathologic lesions seen with sago palm toxicosis in dogs include cirrhosis and marked focal centrolobular and midzonal coagulation necrosis. Secondary CNS lesions may be seen as a result of hepatic encephalopathy and include brain and spinal cord lesions (e.g., demyelination and axonal degeneration).

**TREATMENT**

Treatment of sago palm toxicosis should be directed at preventing further toxicant absorption via decontamination and symptomatic supportive care. In asymptomatic patients—that is, with recent ingestion—aggressive decontamination should be performed. Appropriate emetic agents (for dogs: hydrogen peroxide or apomorphine) should be used if the patient is not already vomiting. Multiple doses of activated charcoal (1-2 g/kg, orally) should be administered, with the first dose containing a cathartic (e.g., sorbitol) if possible. Antiemetics should be promptly initiated (e.g., maropitant, dolasetron, ondansetron) to aid in patient comfort and to prevent emesis of charcoal.

With sago palm toxicosis, fluid therapy is a key part of treatment. Ideally, the use of a balanced crystalloid (e.g., Normosol-R) should be used to aid in hepatic perfusion and treat ongoing fluid losses from vomiting and diarrhea. Lactated Ringer’s Solution (LRS) should be avoided if hepatic injury is evident; with hepatic injury, hyperlactatemia may occur due to inappropriate metabolism of the buffer, lactate, to bicarbonate by the liver. In hypoproteinemic patients (TS < 5.0 g/dL), the use of colloids (e.g., Hetastarch) can be considered to maintain colloid osmotic pressure (COP).

Ancillary treatment for sago palm toxicosis includes the use of benign hepatoprotectants, such as N-acetylcysteine, S-adenosyl methionine (SAMe), and silybin. The mechanism of action of the hepatoprotectant and dosing is listed below:

**N-acetylcysteine**

- Increases glutathione
- Anti-inflammatory
- Improved microcirculation
- May improve oxygen delivery
- Dosing:
  - 280 mg/kg loading dose
  - 70 mg/kg every 6 hours for 7 to 17 doses
  - If normal liver function tests at 48 hours, discontinue

**SAMe**

- Increases glutathione
- Improves membrane stability
- Cytokine modulation
- Anti-apoptotic
- Dosing:
  - 20 mg/kg PO for 30 days

**Silibin (milk thistle)**

- Reactive oxygen species scavenger
• Anti-inflammatory
• Anti-fibrotic
• Increases protein synthesis
• Choleretic
• Dosing: 20–50 mg/kg/day every 6 to 8 to 24 hours PO

Additional treatment includes GI support for presumptive increased gastric levels (e.g., H2 blockers, proton-pump inhibitors, sucralfate), neurologic support (e.g., anticonvulsant therapy, treatment for secondary hepatic encephalopathy), dextrose supplementation, blood pressure monitoring, thermoregulation, and symptomatic supportive care.

In severe cases of AHN with measurable coagulopathy, the use of vitamin K1 (e.g., 1 mg/kg PO or subcutaneously, SC, every 12 hours) and plasma transfusions [as either frozen plasma (FP) or fresh frozen plasma (FFP)] is warranted. Additional treatment may require dextrose supplementation, anticonvulsants, broad-spectrum antibiotic therapy (due to altered portal circulation), and specific treatment for hepatic encephalopathy (e.g., lactulose, neomycin), depending on the severity, extent, and degree of liver injury.

**PROGNOSIS**

Unfortunately, the reported mortality for sago palm toxicosis ranges from 32% to 50%. In recent studies, non-survivors were more likely to have:

• Evidence of increased ALT and total bilirubin
• Evidence of coagulopathy (prolonged PT/PTT)
• Hypoalbuminemia

Survivors were more likely to have received activated charcoal, which reiterates the importance of aggressive decontamination with this plant. Overall, long-term recovery is poor, as the potential for chronic liver disease exists.

**CONCLUSION**

Veterinarians should be aware of the expanding availability of sago palm plants in North America and the dangers of sago palm ingestion resulting in severe, potentially fatal AHN or hepatic injury. Pet owners should be cautioned about the dangers of sago palm plants. The aggressive use of decontamination and symptomatic supportive care is necessary to ensure good outcome.

**REFERENCES**

Megaesophagus and Focal Myasthenia Gravis in Dogs
Todd R. Tams, DVM, DACVIM • David S. Biller, DVM, DACVR

SUMMARY
Megaesophagus is one of the most common causes of regurgitation in dogs and can be congenital or acquired. The acquired form can be idiopathic or caused by a specific syndrome. Failure to recognize an underlying cause can significantly complicate matters and make it more difficult to manage the patient successfully, and can also lead to sudden demise of the patient if catastrophic aspiration pneumonia occurs. Focal myasthenia gravis is one of the known causes of megaesophagus, and it is important that any dog with esophageal weakness be evaluated for focal myasthenia gravis. Many dogs with this potentially devastating disease can be managed well for a number of years by very dedicated pet owners. The emphasis of this article is on the diagnosis and management of the dogs with this condition.

CRITICAL KEYS
• Megaesophagus is one of the most common causes of regurgitation in dogs, and is a frequent finding in dogs with myasthenia gravis (MG).
• Acquired megaesophagus is most commonly diagnosed by the presence of generalized esophageal dilation on survey thoracic radiographs, without evidence of obstruction. Once the presence of megaesophagus is confirmed, appropriate ancillary tests should be performed in search of an underlying cause.
• The diagnostic test of choice for myasthenia gravis is an acetylcholine receptor antibody titer assay, which is run at the Comparative Neuromuscular Laboratory in La Jolla, CA. The assay is specific and sensitive and documents an autoimmune response against acetylcholine (Ach) receptors.
• The main objectives of treatment for regurgitation disorders are to remove the initiating cause as early as possible, minimize chances for aspiration of esophageal content, and maximize nutrient intake to the GI tract.
• Megaesophagus patients are best fed with the upper body in an elevated position of at least 45 degrees (more if possible). A special feeding chair (the Bailey Chair) has been designed for this purpose. The elevated position should be maintained for a full 10 minutes after ingestion of food is completed.
• In the past it was thought that most dogs with megaesophagus had a poor prognosis. Experience has shown, however, that many dogs with this potentially devastating disease can be managed well for a number of years by very dedicated pet owners.
• The cholinesterase inhibitor pyridostigmine bromide is the cornerstone of therapy for myasthenia gravis; it is advised that veterinarians who have not had much experience managing dogs with focal or generalized MG actively consult with a veterinary neurologist or internist.
Megaesophagus is one of the most common causes of regurgitation in dogs. The term megaesophagus refers to a specific syndrome characterized by a dilated hypoperistaltic esophagus and should be differentiated from other causes of esophageal dilation (e.g., foreign body, vascular ring anomaly, mucosal stricture, neoplasia) which may or may not be characterized by abnormal peristalsis. Megaesophagus can be congenital or acquired. Acquired megaesophagus can be idiopathic or caused by a specific syndrome, such as myasthenia gravis, hypoadrenocorticism, lead toxicity, and others. It is always important to perform tests to determine if an underlying disease is present, as successful treatment of the primary disease may in some cases lead to resolution of esophageal hypomotility, either completely or at least partially. Failure to recognize an underlying cause can significantly complicate matters and make it more difficult to manage the patient successfully, and can also lead to sudden demise of the patient if catastrophic aspiration pneumonia occurs.

In the past it was thought that most dogs with megaesophagus had a poor prognosis. Experience has shown, however, that many dogs with this potentially devastating disease can be managed well for a number of years by very dedicated pet owners. The keys to success include identifying and treating any underlying cause and finding a way to successfully provide nutritional support with delivery of nutrients to the stomach, while minimizing episodes of regurgitation since these can lead to aspiration of food to the airways, debilitating pneumonia, and death in some cases. Feeding dogs in a completely upright position using high chairs (e.g., Bailey Chair) has made a significant difference in our ability to manage dogs more successfully for long periods of time.

MEGAESOPHAGUS AND MYASTHENIA GRAVIS

Megaesophagus is a frequent finding in animals with myasthenia gravis (MG). There are two main types of MG in dogs, a generalized form and a focal form. MG can be congenital or acquired. Acquired MG is an immune-mediated disease in which antibodies (in most cases IgG) are formed against the nicotinic acetylcholine (Ach) receptors, resulting in decreased numbers of receptors on the postsynaptic membrane. These autoantibodies can alter the receptor function by one of several mechanisms, and the result is a decrease in normal neuromuscular junction (NMJ) transmission which then results in muscle weakness.

Acquired MG can present as one of three different clinical syndromes: focal MG, generalized MG, and acute fulminating MG. Acquired MG has been associated with other diseases; therefore, when planning the diagnostic approach the clinician should keep these diseases in mind. These diseases include:

- Hypothyroidism and other autoimmune diseases
- Thymomas
- Thymic cysts
- Nonepitheliotropic cutaneous lymphoma
- Cholangiocellular carcinoma
- Anal sac adenocarcinoma
- Osteogenic sarcoma

In dogs, a high risk for MG exists in several breeds, including Akitas, several terrier breeds, German shorthaired pointers, and Chihuahuas. The German shepherd dog and golden retriever demonstrate the highest morbidity.

Generalized MG patients have skeletal muscle weakness that may be induced or exacerbated by exercise as a hallmark sign. In contrast, focal MG presents as a weakness of isolated muscle groups, particularly the esophageal, pharyngeal, laryngeal, and facial muscles. There is an absence of appendicular muscle weakness in focal MG. Acute fulminating MG is a severe and rapidly progressing form of MG.

CLINICAL SIGNS ASSOCIATED WITH MEGAESOPHAGUS

The dominant clinical sign of megaesophagus is regurgitation. It is essential that the clinician make a clear differentiation between regurgitation and vomiting at the outset. Failure to recognize the difference between regurgitation and vomiting often leads to inappropriate testing (i.e., tests most useful for diagnosis of abdominal disorders are generally performed), misdiagnosis, and the use of ineffective treatment regimens. Valuable time is wasted when tests for vomiting rather than regurgitation are pursued. Therefore, the first diagnostic step is to obtain an accurate history and to personally observe the actions of the patient so that an accurate determination of regurgitation versus vomiting can be made.

Regurgitation refers to a passive, sometimes almost effortless retrograde movement of ingested material to a level proximal to the upper esophageal
sphincter. Usually this occurs before ingested material reaches the stomach. There usually are few additional premonitory signs (e.g., retching, signs of nausea) except ptyalism in esophageal inflammatory or obstructive disease. *Vomiting* refers to an active process with forceful ejection of gastric content and often proximal small intestinal contents as well through the mouth. The vomiting act involves three stages: nausea, retching, and vomiting. Regurgitated material does not contain bile, whereas vomited material frequently does. In fact, content of ejected material is an important point of discussion with clients. Dogs with regurgitation often bring up food with foam and saliva, but there is no bile present, unless the dog has both regurgitation and vomiting. Both regurgitation and vomiting are clinical signs of many disorders and should not be considered a primary disease. Regurgitation is usually a clinical sign of an esophageal disorder.

The esophagus is a tremendously dilatable muscular tube that acts via a series of well-coordinated peristaltic contractions to move ingesta from the mouth to the stomach. Regurgitation in most cases results from abnormal esophageal peristalsis, esophageal obstruction, or asynchronous function of the gastroesophageal junction. Significant complications of regurgitation include aspiration pneumonia and chronic wasting disease. All patients with esophageal dysfunction are at risk for sudden death related to aspiration and subsequent upper airway obstruction.

Regurgitation may occur minutes to hours after eating. Frequency varies from several episodes per week to many (10 to 20) episodes in a single day in some patients. It must be recognized that the degree of esophageal function does not always correlate with the severity of clinical signs. Other clinical signs may include acute or chronic cough that may or may not be associated with dyspnea and fever. These signs are most consistent with aspiration pneumonia, which is the most complication of megaesophagus. Coughing may also be related to compression of lung tissue and airways by the enlarged esophagus and its contents. Occasionally coughing is the only clinical sign demonstrated by a dog with megaesophagus. Weight loss and emaciation occur secondary to inadequate food intake. Inappetence or salivation of both may result from discomfort caused by esophagitis.

In patients in which megaesophagus is associated with an underlying disorder, other clinical abnormalities that may be noted include:

- Generalized muscle weakness (generalized MG, polymyopathy, hypoadrenocorticism)
- Neurologic deficits (MG, central nervous system disease, polyneuropathy)
- Generalized muscle atrophy or pain with myositis
- Vomiting (hypoadrenocorticism, lead poisoning, obesity, and alopecia with hypothyroidism)
- Oropharyngeal dysphagia with generalized neuromuscular dysfunction

**DIAGNOSIS**

Acquired megaesophagus is most commonly diagnosed by the presence of generalized esophageal dilation on survey thoracic radiographs, without evidence of obstruction. Once the presence of megaesophagus is confirmed, appropriate ancillary tests should be performed in search of an underlying cause (e.g., MG, hypoadrenocorticism). Details are described in the following sections.

**Diagnostic Imaging**

**Survey Radiographs**

The normal esophagus is not often seen in the canine patient, although a small amount of fluid may accumulate within the caudal thoracic esophagus creating a soft tissue tubular-appearing structure midway between the caudal vena cava and aorta, especially in thin dogs positioned in left lateral recumbency. A small amount of gas within the esophageal lumen, especially caudal to the cranial esophageal sphincter, at the level of the thoracic inlet, and dorsal to the heart may be within normal limits. The inability to visualize the esophagus on thoracic images does not rule out esophageal disease. One unusual situation to be aware of is that in cases where animals are severely dyspneic (e.g., with severe pneumonia) the esophagus may become dilated with gas. If this is indeed a secondary problem, then the megaesophagus will also resolve with resolution of the dyspnea.

**Contrast Study: Esophagram**

The esophagram is the positive esophageal contrast study necessary for further characterization and evaluation of almost all cases of morphologic and dynamic esophageal diseases. Dynamic or functional disease may
need sequential images with contrast or fluoroscopy. Sedation should be avoided as it may affect esophageal motility.

**Technique:** An esophagram is done using either barium liquid or barium paste. These products provide both great radiopacity and may demonstrate adherence to the mucosa of the esophagus (barium paste does a much better job of this). In some cases where animals have problems swallowing solids and not liquids or have a suspected esophageal stricture or dysfunction barium may be mixed with either canned or dry dog food.

A survey radiograph should always be taken prior to any contrast study. The entire esophagus should be evaluated on survey images including from the proximal esophagus to the junction with the stomach. The esophagus is a midline structure and will remain superimposed with spine and sternum on ventrodorsal (VD) images; therefore an oblique 15- to 30-degree VD view should be utilized instead of an orthogonal view to lateral image. Approximately 10 to 30 mL of contrast should be given orally to induce swallowing and potentially coat the esophagus before radiographs are taken (enough time should be given to allow contrast to fully pass to stomach). This can be repeated several times if necessary.

**Normal Appearance of Esophagus on Esophagram:** The canine esophagus normally appears as a series of parallel longitudinal folds. Between these folds there may be small thin linear accumulations of positive contrast. On VD or VD oblique images the esophagus will pass to the left of the trachea at the level of the thoracic inlet. If a bolus of contrast is seen on esophagram a subsequent image should be taken to make sure it is not present on multiple images.

There are a few variations in the normal appearance of the esophagus that you should recognize. The first one is a small amount of air in the cervical esophagus, just caudal to the cricopharyngeus muscle. The second variation of normal is a small thin pocket of air in the thoracic esophagus, just cranial to the heart base. With a three-view thoracic series, there will often be some fluid in the caudal esophagus on the left lateral projection. This is most likely because the esophagus and cardia of the stomach are on the left, and the increased pressure from abdominal organs causes some reflux of gastric contents. The key to recognizing all of these variations of normal is that they are transient. If another radiograph is performed, they should be gone (see description of technique above).

**Radiographic Changes with Megaesophagus**

Definitive diagnosis of megaesophagus requires the identification of a dilated esophagus on radiographs. In some cases survey radiographs may demonstrate the etiology of the esophageal dilation [e.g., foreign body, mass lesion, hiatal hernia, gastric dilatation-volvulus (GDV)]. Megaesophagus is one of those conditions that covers a broad range of severity. Radiographs may show an esophagus dilated with air, fluid, ingesta/foreign material, or a mixture of these. The esophagus can have a mild or severe, focal, segmental/regional or generalized dilation. It is also supportive if there is more than one radiograph demonstrating persistent dilation.

If megaesophagus is diagnosed on survey radiographs an esophagram is generally not necessary. Some believe that an esophagram may pose a significant risk of aspiration especially with barium contrast that pools in the esophagus. Evaluation of lungs for aspiration pneumonia as a common sequela of megaesophagus is also of utmost importance.

**Esophagram/Barium Swallow Changes with Megaesophagus**

If the patient has signs of a swallowing disorder or regurgitation, and megaesophagus is suspected but not appreciated on survey images, administering barium contrast and taking a radiograph will be enough to demonstrate the outline of the dilated esophagus (see description of technique below). Fluoroscopy or sequential images are very valuable in more subtle cases since the motility in real time or over time can be observed. Once the diagnosis of megaesophagus has been made, additional radiographs or fluoroscopy may be indicated in monitoring response to treatment, or for response to treatment of secondary disease such as aspiration pneumonia.

**Laboratory Tests**

A baseline complete blood count (CBC) and biochemical profile should be run in all patients with megaesophagus to look for evidence of underlying problems. Specific tests to evaluate for systemic disorders such as hypoadrenocorticism [adrenocorticotropic hormone (ACTH) stimulation], systemic lupus erythematosis (antinuclear antibody), and serum lead levels are done if the history and/or physical examination indicate that these primary disorders may exist. Focal myasthenia gravis should be considered in any patient with megaesophagus. The test of choice is
an acetylcholine receptor antibody titer assay. The assay is specific and sensitive and documents an autoimmune response against acetylcholine (Ach) receptors. The acetylcholine receptor antibody test is run at the Comparative Neuromuscular Laboratory in La Jolla, CA. This laboratory is headed by a veterinary internist, Dr. Diane Shelton, who is highly experienced in diagnosis and management of neuromuscular disorders. The laboratory is an international reference center dedicated to the diagnosis and study of spontaneous neuromuscular diseases in companion animals. Contact the laboratory for forms, sample submission instructions, and interpretation of results. The address is:

**Comparative Neuromuscular Laboratory**
9500 Gilman Drive
Basic Science Building, Rm. 2095
University of California, San Diego
La Jolla, CA 92093-0709
Phone: (858)534-1537 Fax: (858)534-0391
Web: http://vetneuromuscular.ucsd.edu/
Email: musclelab@ucsd.edu

An Ach receptor antibody concentration >0.6 nmol/L is positive for dogs. False-positive tests are extremely rare and therefore a positive result is considered virtually definitive for acquired MG. The serum Ach receptor antibody concentration is usually lowest in dogs with focal MG and highest on dogs with fulminant MG.

**REPEAT TESTING MAY BE NECESSARY:** In some cases the ACh receptor antibody titer test may be within normal reference range on initial testing, but then may be abnormal on recheck testing performed 2 to 3 months later. VERY IMPORTANT: Clinicians should retest any patient that is still considered to possibly have focal MG. If a subsequent test is positive, then there is clear direction at that time on how to manage the patient more specifically in addition to using elevated feedings for megaesophagus (i.e., use drug therapy for MG).

**TREATMENT**

**General Management Principles for Megaesophagus**

The main objectives of treatment for regurgitation disorders are to remove the initiating cause as early as possible, minimize chances for aspiration of esophageal content, and maximize nutrient intake to the GI tract. In most cases, idiopathic megaesophagus is incurable, and treatment involves an individually tailored feeding regimen with the patient eating in an elevated position. Medical management is indicated for such secondary causes of esophageal dilation as myasthenia gravis, hypoadrenocorticism, hypothyroidism (a rare cause of megaesophagus), lead toxicity, and systemic lupus erythematosus. Any occurrences of aspiration pneumonia should be treated aggressively.

Megaesophagus patients are best fed with the upper body in an elevated position of at least 45 degrees (more if possible). It is important that proper positioning be clearly demonstrated to the client so that there is no misunderstanding. The elevated position should be maintained for a full 10 minutes after ingestion of food is completed. Various props to aid in the elevation process have been used successfully, including ladders, stairs, ramps, tables, and chairs. Since the esophagus is virtually never completely empty in a megaesophagus patient it is often helpful to hold the animal in an elevated position for 5 to 10 minutes at a time sometime between meals and at bedtime.

**A Recent Advance: Use of Special Feeding Chairs**

**Bailey Chair:** There is a lot of information available on the internet about a special feeding chair that was designed by Donna and Joe Koch, the owners of a dog named Bailey, who had been diagnosed with megaesophagus. The dog sits in a totally upright (“begging”) position to eat, drink, or take medication and gravity aids transit of anything ingested to the stomach. Basically we are turning the patient into a biped from a quadruped. Use key words “Bailey Chair” for an internet search and information on how to acquire or build a Bailey Chair (also see support group information below).

Megaesophagus patients are ideally fed two to four times daily. This depends, of course, on the caregiver’s time constraints. We have had the best success feeding soft moist to solid (chopped) canned food consistency. We only recommend trying gruels if the semi-moist consistency is not well tolerated. Some patients do well when fed a series of “meatballs” fashioned from canned food. Others can tolerate dry food fairly well. A key point is that each patient is an individual and clients should be instructed to experiment with various food consistencies in order to determine the best approach for their own pet.

Many patients with idiopathic megaesophagus can be managed
successfully for months to years. We have known many dedicated owners who have managed to find the time required to care for their pets. As a result of this experience we try to offer as much encouragement as possible at the time of diagnosis. The most worrisome complications that can occur are aspiration pneumonia and significant weight loss. The prognosis is guarded to poor in patients that suffer recurrent episodes of pneumonia.

**Gastric Feeding Tube Placement**

An option in cases where frequent regurgitation remains an ongoing problem with or without aspiration events is to place a gastric feeding tube [e.g., percutaneous endoscopy-guided gastrostomy tube (PEG)]. All food and water can then be administered through the feeding tube; some patients have been maintained for as long as 4 or more years in this way. Periodic tube replacement will be necessary. Low profile feeding tubes often work best for long-term tube feeding.

**Megaesophagus Client Information and Support Group**

- **www.caninemegaesophagus.org** - Provides information on the causes of congenital and idiopathic canine megaesophagus, the clinical signs, risk factors and accompanying disorders, with lots of feeding tips.
- **A Support Group for owners of dogs that have, had or may have megaesophagus, was established at Yahoo Groups in 2002 by Dave Kay and Katy Weeks, in memory of their golden retriever, Rusty. Members of the group can provide suggestions and ideas for feeding and care of dogs with megaesophagus. A veterinarian monitors the group as an advisor and offers suggestions for members to discuss with their veterinarians. The group is at: http://groups.yahoo.com/group/megaesophagus/ and requires membership.

**Medical Management of Focal Myasthenia Gravis**

Each patient needs to be carefully assessed and an individual treatment plan developed. With an accurate diagnosis and early treatment, autoimmune MG is a treatable disease. It is not uncommon for dogs with MG, unlike humans, to go into spontaneous remission. Treatment duration can range from several months to several years, depending on the severity of disease. Drug therapies can include cholinesterase inhibitors (cornerstone of therapy and recommended for all dogs with MG), corticosteroids, and other immunosuppressive drugs. It is advised that veterinarians who have not had much experience managing dogs with focal or generalized MG actively consult with veterinary neurologists or internists who have much more experience in this area, so that each patient will have the benefit of the expertise needed to navigate the decisions on which drugs to use and when to make adjustments.

Anticholinesterase drugs prolong the availability of Ach for binding to Ach receptors by inhibiting degradation of acetylcholinesterase. **Pyridostigmine bromide** (Mestinon) is administered to dogs at 0.5 to 3 mg/kg PO every 8 to 12 hours. Start low and titrate upwards gradually to achieve the best clinical response while avoiding cholinergic side effects, which can include hypersalivation, vomiting, and muscle fasciculations based on response. Adverse effects may be reduced by administering small doses of atropine or giving the medication after meals when feasible. Tablet and syrup forms are available. The syrup formulation should be diluted 50:50 in water because gastric irritation may result if it is given straight. The response to anticholinesterase therapy can be variable depending on the patient, with some responding significantly and others minimally. Also, anticholinesterase therapy seems to exert greater effect on improving appendicular muscle weakness (in generalized MG) than it does on improving esophageal function in dogs with megaesophagus.

Use of immunosuppressive therapy in dogs with acquired MG is based on the underlying pathophysiology, which includes an autoimmune destruction of functional Ach receptors. However, there can be concerns related to use of immunosuppressive therapy, including immunosuppressing dogs with aspiration pneumonia, a common sequela of megaesophagus. Glucocorticoid therapy can also cause a worsening of muscular weakness. Further, the increased water consumption associated with steroid use can be problematic in dogs with swallowing difficulties or that are regurgitating. Therefore, the decision to use steroids should be made carefully with consideration of the entire clinical picture, and steroids are withheld until pneumonia is resolved. In some cases it may be best to first try pyridostigmine and if there is a suboptimal response steroids are initiated at a conservative dose.

When using corticosteroids, it may be best to start with a lower anti-inflammatory level dose of prednisolone, e.g., 0.5 mg/kg given every 24 hours. Beneficial effects of steroids may be related to inhibitory effects of prednisone on the formation and release of inflammatory agents, lymphocyte division, lymphocyte reactivity to Ach receptors, and leukocyte chemotaxis.
Other immunosuppressive drugs that may be beneficial include azathioprine, cyclophosphamide, cyclosporine, and mycophenolate mofetil (MMF). Controlled clinical trials are needed to help determine which drugs are the most effective and what the best protocols are for dogs that require more than pyridostigmine and steroids.

**Azathioprine** has been the most commonly used immunosuppressive agent for acquired MG, after prednisolone. Bone marrow suppression is the most common adverse event, and occasionally pancreatitis can occur as well. The recommended dose is 1.1 to 2.2 mg/kg once every 24 hours. Alternate-day therapy is given later when the patient is well controlled. There is a lag phase of 2 to 4 weeks. Azathioprine allows for lowering of the steroid dose once it has reached therapeutic levels (“steroid sparing” effect). A complete blood count should be run 7 to 10 days after azathioprine is initiated and then monthly thereafter for the first 3 to 4 months.

**Mycophenolate mofetil** has been widely used in human medicine. It is being used more commonly now in veterinary medicine but studies are needed to further define indications and effectiveness in dogs.

### Monitoring Dogs with Acquired MG

There is value in monitoring the Ach receptor antibody titer test, especially in dogs that are not on immunosuppressive therapy. Therapy should be continued as long as the test remains positive. Dr. Diane Shelton has reported that at her laboratory there has been an excellent correlation between resolution of clinical signs, including megaesophagus, and return of Ach receptor antibody titers to within the reference range. Once remission occurs, recurrence of MG is rare. **Caution:** A decrease in the Ach receptor antibody titer in dogs that are on immunosuppressive therapy and that are no longer demonstrating clinical signs should not be interpreted as a sign of remission. It is known that once the immunosuppressive drug dosage is decreased clinical signs can return.

### REFERENCES


Feline Esophageal Stricture
David C. Twedt, DVM, DACVIM • David S. Biller, DVM, DACVR • Howard B. Seim III, DVM, DACVS

SUMMARY
Esophageal stricture is the result of trauma to the esophageal wall from acid, foreign bodies, or medications, most notably doxycycline. The diagnosis requires appropriate imaging studies and/or endoscopy. The management of these strictures in cats is difficult, with serious potential complications. Treatment involves either feeding a liquid diet or therapy using balloon dilation. Prognosis is generally good but repeated dilations may be required at frequent intervals to maintain luminal diameter.

CRITICAL KEYS
• Esophageal stricture is the result of trauma to the esophageal wall from acid, foreign bodies, or medications, most notably doxycycline. The diagnosis requires appropriate imaging studies and/or endoscopy. The management of these strictures in cats is difficult, with serious potential complications. Treatment involves either feeding a liquid diet or therapy using balloon dilation. Prognosis is generally good but repeated dilations may be required at frequent intervals to maintain luminal diameter.
• Regurgitation is the passive expulsion of esophageal contents without forceful retching. In some cases it may be difficult to differentiate regurgitation from vomiting, necessitating investigation of both problems.
• Cats with obstructions from strictures classically regurgitate solid food but are often able to hold down liquids. The severity of clinical signs of regurgitation is related to the degree of esophageal narrowing.
• Benign inflammatory esophageal strictures result following deep submucosal ulceration with subsequent fibrosis, which may be caused by anesthesia-related gastric reflux, foreign bodies, pills, trauma, or esophageal tube placement.
• Doxycycline tablets are reported to be associated with esophageal strictures. Strictures develop due to the failure of pills to pass into the stomach in a timely manner, resulting in local caustic ulceration.
• Routine thoracic radiographs may fail to demonstrate abnormalities and contrast imaging is required. Focal strictures are best demonstrated when a food–barium mixture is given.
• Balloon dilation of strictures generally requires repeated dilations at frequent time intervals (days to weekly) because of the tendency for them to re-stricture. The dilation process can be observed endoscopically to watch for tearing or damage.
• Surgical resection of a stricture is not recommended because of the high rate of re-stricture formation at the surgical site.
• Generally focal esophageal strictures have a good prognosis but repeated dilations may be required at frequent intervals to maintain esophageal luminal diameter.
Esophageal disease is associated with inflammatory conditions, motility disorders, or obstructive disorders. Obstructive disorders of the esophagus include such conditions as inflammatory strictures, foreign bodies, vascular ring anomalies, and neoplasia. Neoplasia is uncommon in dogs and cats; however, we reported on leiomyomas of the distal esophageal sphincter of dogs causing a mechanical obstruction. The two most common causes of esophageal obstruction in the cat are esophageal strictures and esophageal foreign bodies. A definitive diagnosis of an esophageal stricture involves imaging, endoscopy, or both.

Benign inflammatory esophageal strictures result following deep submucosal ulceration with subsequent fibrosis. For strictures to develop there must generally be greater than 180 degrees of submucosal involvement; focal ulceration is less likely to result in a stricture. In a review of 23 cases of strictures anesthesia-related gastric reflux occurred in 65% of the cases while 9% of the cases were associated with foreign bodies and the remainder were due to other causes such as pill-associated, trauma, or esophageal tube placement. The association of gastroesophageal reflux during anesthesia is reported to occur in approximately 10% to 15% of dogs having anesthesia; in most cases reffractory esophagitis may result but rarely in some cases stricture formation occurs. The time from anesthesia to stricture formation is approximately 1 to 2 weeks. Doxycycline, clindamycin, and nonsteroidal anti-inflammatory drugs (NSAIDs) are the most common drugs to cause strictures in humans and we reported a number of cats developing esophageal strictures secondary to doxycycline and clindamycin tablets. The pill-associated strictures occur predominantly in the cervical esophagus. Studies in our laboratory showed cats given pills alone had delayed passage through the esophagus, with retention of many pills in the cervical esophagus. When pill administration was followed by 3 to 6 mL of water, however, the pills usually passed rapidly into the stomach. It is likely in cases in which the doxycycline tablet failed to leave the esophagus, local esophagitis and then stricture formation resulted. It is suspected that drug-associated strictures could also occur in dogs but because pills are often given to dogs with a treat or food, delayed passage through the esophagus is less likely.

Animals with obstructions from strictures classically regurgitate solid food but often are able to hold down liquids. The severity of clinical signs of regurgitation is related to the degree of esophageal narrowing. Mild stricture formation may cause minimal signs unless the animal swallows a large food bolus. The regurgitation usually occurs immediately or shortly following eating. Obstructions more proximal in the esophagus tend to cause immediate regurgitation while more distal lesions tend to retain the food longer because of more luminal space, resulting in delayed postprandial regurgitation. Liquid barium contrast studies may fail to detect strictures and any liquid barium contrast study should be followed by a barium-food mixture esophagram (see below). Endoscopy will confirm the presence and location of the stricture.

**IMAGING**

**Survey Radiographs**

Survey radiographs of the thorax and neck is the first step for imaging cats with suspected esophageal disease. The normal esophagus is usually not

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**CRITICAL KEYS continued**

- Megaesophagus patients are best fed with the upper body in an elevated position of at least 45 degrees (more if possible). A special feeding chair (the Bailey Chair) has been designed for this purpose. The elevated position should be maintained for a full 10 minutes after ingestion of food is completed.
- In the past it was thought that most dogs with megaesophagus had a poor prognosis. Experience has shown, however, that many dogs with this potentially devastating disease can be managed well for a number of years by very dedicated pet owners.
- The cholinesterase inhibitor pyridostigmine bromide is the cornerstone of therapy for myasthenia gravis; it is advised that veterinarians who have not had much experience managing dogs with focal or generalized MG actively consult with a veterinary neurologist or internist.
seen in the feline patient although a small amount of fluid may accumulate within the caudal thoracic esophagus creating a soft tissue tubular appearing structure midway between the caudal vena cava and aorta in lateral recumbency. Survey thoracic radiographs are usually normal, but the inability to visualize the esophagus on thoracic images does not rule out esophageal disease.

While survey images rarely provide diagnosis of cats with esophageal stricture, depending upon the severity and chronicity, the presence of a pre-stenotic dilation (fluid-filled esophagus or a dilated esophagus with fluid and gas) secondary to an esophageal stricture may be visualized. Survey radiographs will also aid in the diagnosis of aspiration pneumonia a common sequel of esophageal strictures.

A survey radiograph should always be taken prior to any contrast study. The entire esophagus should be evaluated on survey images including from the proximal esophagus to the junction of esophagus with stomach. Since the esophagus is a midline structure and will remain superimposed with spine and sternum on ventrodorsal (VD) images, an oblique 15- to 30-degree VD may be utilized as an extra view with lateral and dorsoventral (DV) views. Approximately 5 to 15 mL of barium contrast (30–60% weight-volume) should be given orally to induce swallowing and potentially coat the esophagus before radiographs are taken (enough time should be given to allow contrast to fully pass to stomach). This can be repeated several times if necessary.

Contrast Studies
A contrast esophagram is more sensitive than survey radiographs for defining origins of suspect esophageal disease, and detecting esophageal obstructive lesions. The esophagram is the positive esophageal contrast study necessary for further characterization and evaluation of almost all cases of morphologic and dynamic esophageal diseases. Dynamic or functional disease may need sequential images with contrast or fluoroscopy. Sedation should be avoided as it may affect esophageal motility. An esophagram is necessary to confirm the diagnosis of stricture, and is useful for determining the number, location, and length of strictures. Most animals have a single esophageal stricture.

An esophagram is done using either barium liquid or barium paste. These products both provide great radiopacity and may demonstrate adherence to the mucosa of the esophagus (barium paste does much better job of this). Strictures can be missed with liquid barium alone, but always start with liquid. In some cases in which animals have problems swallowing solids and not liquids or have a suspected esophageal stricture not demonstrated with liquid barium, barium may be mixed with either canned or dry food. Multiple swallows of contrast with subsequent images are sometimes necessary to demonstrate persistent narrowing versus esophageal spasm.

The esophagus normally appears as a series of parallel longitudinal folds through the proximal two thirds of its length. Between these folds there may be small, thin linear accumulations of positive contrast. The caudal third is composed of smooth muscle and with barium contrast appears as a “herringbone” pattern due to the vertical orientation (relative to the long axis of the esophagus) of the striations. On VD or DV oblique images the esophagus will pass to the left of the trachea at the level of the thoracic inlet. If a bolus of contrast is seen on an esophagram a subsequent image should be taken to make sure it is not present on multiple images.

ENDOSCOPY
The endoscopic findings with an esophageal stricture are generally obvious. In cats pill-associated strictures are often located in the cervical region and acid reflux-associated strictures generally occur in the distal esophagus. Often there is evidence of esophagitis and ulceration cranial to the stricture due to failure to pass food. One should be aware that additional strictures caudal to the first stricture may be present and can only be identified once the first stricture is dilated and the endoscope passed distally. The strictures can be ulcerated and irregular but most often they are smooth fibrous constrictions narrowing the esophagus.

TREATMENT
Management for esophageal strictures involves either feeding a liquid diet or therapy using balloon dilation. Surgical resection of a stricture is not recommended because of the high rate of re-stricture formation at the surgical site. It is advised to refer cases to someone having the equipment and expertise to handle the strictures.

Balloon Dilation
The technique involves placing specialized balloons into the stricture and radially dilating the lumen under pressure. Special injection guns are attached to the balloon to dilate at a prescribed pressure. We generally
dilate three times for 60 seconds with increasing sized balloons, repeating
the procedure until the luminal diameter is approximately its normal size
or until there is significant mucosal tearing. In some cases a guide wire
must be placed through the stricture to direct the balloons through if it
is a very small opening. The author (D.T.) generally watches the dilation
endoscopically although some will dilate strictures using fluoroscopic
control, and believes the ability to observe for tearing or damage makes
endoscopic dilation the preferred technique. As part of the therapy we are
also injecting 0.1 mL of triamcinolone (10 mg/mL) using an endoscopic
injection needle in three to four sites submucosally directly into the
stricture prior to dilation. The intent is to reduce local inflammation
following dilation that can result in subsequent re-stricture formation.
In a review of 23 cases 84% were considered to have a good outcome
following a mean of three separate balloon dilations performed at weekly
intervals. The number of dilations, however, could be as few as one or
as many as 10 or more. Doxycycline strictures tend to respond quite
favorably in less than three weekly dilations. In severe cases or animals
suffering from malnutrition it would be wise to place a gastric feeding
tube. If balloon dilation fails to resolve the stricture, stent placement may
be a consideration. Post-dilation management includes feeding soft food,
sucralfate acid blockers, and a prokinetic agent such as cisapride if reflux is
suspected.

REFERENCES AND SUGGESTED READING
Acute Pancreatitis in Dogs – To Feed or Not to Feed
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SUMMARY
Acute pancreatitis in dogs can vary in severity, ranging from mild signs to those cases that are fulminant and frequently fatal, and often results in multiple organ system involvement. Confusion and controversy exist regarding the pathogenesis, diagnosis, and treatment of acute pancreatitis in dogs. There have been many changes in the treatment recommendations for management of acute pancreatitis in recent years. This article focuses on best treatment plans for success, including diagnostics, supportive medical care, feeding recommendations, feeding tube placement techniques, and potential emerging therapies.

CRITICAL KEYS
• Case presentation, serum pancreatic lipase immunoreactivity (PLI, quantitated), and imaging studies are essential components of the clinical diagnosis of acute pancreatitis.
• The diagnosis of pancreatitis cannot be made based on amylase and/or lipase values.
• Liver enzymes are frequently abnormal in dogs with pancreatitis.
• Ultrasonography can provide valuable diagnostic information in most animals with inflammatory or neoplastic diseases of the pancreas if it is done properly and with patience.
• Fluids, pain management, antiemetics, and early feeding are the most important components of initial therapy in patients with acute pancreatitis.
• Maropitant (Cerenia) is very effective for management of vomiting and is a useful adjunct to opioids for control of visceral pain.
• Early enteral feeding is essential in the recovery of pancreatitis. Feeding should be initiated by the second to third day of hospitalization, or earlier as soon as the vomiting is fairly well controlled.
• Recent studies have shown that prepyloric feeding is well tolerated in patients with pancreatitis. Nasoesophageal or esophagostomy tube feeding is initiated for patients who will not eat voluntarily. Low fat nutrients are provided; vanilla Ensure is low in fat and is the liquid diet of choice.
• Hyperbaric oxygen therapy has been shown to be beneficial in management of humans with acute necrotizing pancreatitis and this modality is being used in dogs now as well.
• Acute pancreatitis is rarely a surgical disease but surgery may be indicated with evidence of bacterial peritonitis or mass.
Pancreatitis is a common disorder of dogs but due to challenges in establishing a definitive diagnosis the true incidence of pancreatitis seen in clinical practice is not known. Mild cases of pancreatitis that do not show “classical” signs such as acute vomiting and abdominal pain can be very difficult to diagnose. Conversely, pancreatitis is just one of many causes of vomiting and clinicians are challenged daily to try to determine a specific cause in patients presented with vomiting and other clinical signs. Most dogs have acute pancreatitis and most cats have chronic pancreatitis. Clinical presentation and diagnostic criteria differ between species and type of pancreatitis. Acute pancreatitis is potentially reversible but can also be fatal while chronic pancreatitis generally has irreversible changes but is rarely fatal.

Confusion and controversy exist regarding the pathogenesis, diagnosis, and treatment of acute pancreatitis in the dog. The spectrum of clinical disease can range from mild signs to those that are fulminant and frequently fatal. It is the development of multisystemic abnormalities that separates mild from severe, potentially fatal pancreatitis. Pancreatitis can be broadly categorized as acute, recurrent acute, or chronic. Medical management is the main form of treatment in most cases. Surgical intervention in diagnosis and management of pancreatitis is not often undertaken and is generally reserved for complex cases of pancreatitis, such as acute necrotizing pancreatitis, pancreatic abscess, pancreatic pseudocyst, and generalized peritonitis secondary to pancreatitis.

ETIOLOGY

The etiology of pancreatitis is generally unknown. Factors that influence development of pancreatitis include high fat diet, obesity, hyperlipoproteinemia, drugs (e.g., thiazides, furosemide, tetracycline, L-asparaginase, azathioprine, corticosteroids), duodenal reflux into pancreatic ducts, pancreatic duct obstruction (e.g., parasites, calculi, neoplasia, inflammation, surgery), hypercalcemia, trauma, and pancreatic ischemia [e.g., shock, gastric dilatation–volvulus, (GDV)].

RISK FACTORS

Risk factors identified in patients with acute pancreatitis include breed (Yorkshire and Silky terrier, toy poodle, miniature Schnauzer, nonsporting breeds), overweight body condition, small breed size, prior gastrointestinal disease, ingestion of garbage and table scraps, diabetes mellitus, hyperadrenocorticism, hypothyroidism, and a history of surgery within 2 weeks prior to development of pancreatitis.

DIAGNOSIS

Acute pancreatitis is one of the most difficult diseases to diagnose. There is no one definitive diagnostic test for pancreatitis, except for histopathology. Biopsy procedures, however, are not commonly done in dogs with suspected acute pancreatitis. Clinical diagnosis is based on a combination of history, physical examination, and compatible clinicopathologic and imaging findings. The clinician’s index of suspicion is important and keeping an open mind to various possibilities is essential (i.e., avoid “tunnel vision”), since multiple organs can be involved concurrently. When vomiting is associated with systemic signs or is persistent the clinician must differentiate metabolic, polysystemic, toxic, and infectious causes from abdominal causes. Pancreatitis can be “diagnosed” when it is not actually present and easily missed when it is present. Most patients with suspected pancreatitis are managed based on the patient’s condition (mild versus severe signs) and the various test results and closely monitoring response to therapy.

Laboratory Findings

Laboratory findings in dogs with pancreatitis are quite variable and to some extent parallel the severity of the clinical disease and may be representative of multiple organ system involvement in patients with severe pancreatitis. Laboratory results might include leukocytosis, azotemia (prerenal and renal), increases in alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP), icterus, hyperglycemia, hypokalemia, acid base changes, disseminated intravascular coagulation (DIC), and increases in amylase, lipase, concentration of trypsin-like immunoreactivity (cTLI), and pancreatic lipase immunoactivity (cPLI).

Hematologic findings are quite variable, ranging from mild neutrophilia and slightly increased hematocrit (dehydration) to marked leukocytosis with a left shift, leukopenia with a degenerative left shift, and anemia. However, some dogs with even moderate to severe pancreatitis can have a normal leukogram. Platelet numbers should be assessed. If there is thrombocytopenia, tests for DIC should be performed [one-stage prothrombin time (OSPT), activated partial thromboplastin time (APTT),
fibrin degradation products (FDP or D-Dimer), and antithrombin III.

Urinalysis allows azotemia to be characterized as prerenal or renal and provides key information for ruling-in or out other disorders, such as diabetes mellitus (glucosuria or ketonuria), and pyelonephritis, which can cause vomiting and abdominal pain (absence of white cell casts or bacteria helps rule out).

Serum amylase and lipase activities are insensitive and nonspecific for pancreatitis. Specificity for both tests is only around 50%. Amylase and lipase are found in the oral cavity, GI tract, pancreas, and liver. Dogs with gastritis or intestinal foreign bodies can have a significantly elevated lipase but no gross evidence of pancreatitis. Lipase is produced by the gastric mucosa, which explains why it can be increased in disorders of gastric inflammation. Increases in amylase and lipase can be seen in renal disease as well (decreased clearance). Significantly increased amylase or lipase can certainly suggest the possibility of pancreatitis, but some dogs with even severe pancreatitis have demonstrated normal levels. All things considered, amylase and lipase assessment are not very useful in the diagnosis of pancreatitis.

Clinical factors that help support a diagnosis of pancreatitis include high index of suspicion, acute or chronic vomiting, weakness, abdominal pain, dehydration, diarrhea, fever, and shock. There may be a history of recent dietary indiscretion or drug administration. There is no apparent sex predilection. Middle-aged to older dogs (greater than 5 years) that are overweight are at higher risk.

Currently, the combination of a quantitated cPLI assay and ultrasonography are considered to provide the most meaningful noninvasive diagnostic information in the clinical setting for determination of whether or not pancreatitis is present. The immunoreactive canine pancreatic lipase assay (cPLI) is the most sensitive assay currently available. Use of the immunoassay allows for the specific measurement of lipase originating from the exocrine pancreas. The sensitivity for serum cPLI concentration is over 85%. However, the specificity is around 75%, meaning that around one fourth of animals without clinical pancreatitis may have a positive cPLI assay. Test interpretation is as follows:

- If the cPLI is > 200 µg/L, pancreatitis should be included in the differential diagnosis and the diagnosis is confirmed through other diagnostics.

- If the cPLI is normal, the patient does not likely have pancreatitis and the clinician should continue to look for another disease.

If pancreatitis is unlikely, it is not warranted to run a PLI as false-positive results are possible. cPLI can be used for diagnosing pancreatitis in dogs with renal failure. A study has shown that, while cPLI is elevated in dogs with renal failure compared with levels in healthy dogs, it is usually still within the reference range (in the higher end of the reference range), but not above the currently recommended cutoff value for pancreatitis. Also, the long-term oral administration of prednisone does not affect cPLI values.

Radiology

The pancreas is shaped like a boomerang, with three parts: the right lobe, which lies adjacent to the descending duodenum; the body, which lies at the junction between the pylorus and duodenum; and the left lobe, which lies adjacent to the greater curvature of the stomach. The pancreas is not normally seen.

With a diseased pancreas, the duodenum may be displaced ventrally. On the ventrodorsal view, there is usually lateral (right) displacement of the descending duodenum and the pylorus is displaced to the left (widening of the duodenal pyloric angle). The transverse colon as well as ascending colon may be displaced centrally and caudally. Fluid and gas distention of the stomach, duodenum, and colon may be present. Localized loss of abdominal detail (ground-glass appearance) may occur with inflammation of the pancreas. The most common change radiographically on survey films is actually no change at all.

Changes that may occur and be visualized relative to pancreatic disease and an upper GI series include fixed position and shape to the duodenum; widened proximal duodenal flexure (the angle between the pylorus and duodenum); and thickening and rigidity of the duodenum, pylorus, and greater curvature of the stomach. Gastric outflow obstruction and duodenal distention (ileus) may also be visualized.

Ultrasonography

Ultrasonography is the imaging method of choice for evaluation of the pancreas in small animals. It can provide information about the size, shape, and contour of the pancreas and may suggest the presence of inflammation, abscess formation, or neoplasia. Ideally, patients should be
fasted before abdominal ultrasonography to minimize interference from GI gas.

There are several limitations to pancreatic ultrasonography:

• The normal pancreas is not always seen as a discrete structure; thus, it is actually the pancreatic area, and not the organ itself, that must be examined.

• Ultrasonography lacks specificity. For the most part, ultrasonographic findings do not allow differentiation between inflammatory and neoplastic processes.

• The proximity of the pancreas to gas in the stomach, colon, and duodenum may prevent complete and accurate evaluation of the pancreatic region.

Despite these disadvantages, ultrasonography can provide valuable diagnostic information in most animals with inflammatory or neoplastic diseases of the pancreas if it is done properly and with patience. The healthy pancreas is difficult to image as a distinct organ. Therefore, familiarity with the anatomy of adjacent structures is crucial to successful evaluation of the pancreatic area.

The left lobe of the pancreas is dorsocaudal to the stomach and dorsocranial to the transverse colon. Its distal aspect can be visualized cranial to the left kidney and medial to the spleen. The pancreatic body lies caudal to the pylorus. It is ventral to the portal vein and the caudate process of the liver and craniodorsal to the right kidney. The right lobe of the pancreas is found dorsomedial to the descending duodenum, ventral to the right kidney, and ventrolateral to the portal vein. Both the cranial and caudal pancreatoduodenal veins are located in the parenchyma of the right lobe and run parallel to the descending duodenum.

Normally, pancreatic parenchyma has a homogeneous echotexture. The pancreaticoduodenal vein may be apparent in the right lobe. The left lobe of the healthy pancreas occasionally is seen in the triangular region defined by the spleen, stomach, and left kidney. Gas in the adjacent stomach and transverse colon makes imaging of all but the most distal portion of the left lobe difficult.

Ultrasonography Findings in Pancreatitis

Ultrasonography has proven to be a reliable tool for identifying changes associated with pancreatic inflammation and is currently considered the imaging method of choice for evaluating pancreatitis and pancreatic neoplasia. Ultrasonography provides several distinct advantages in the diagnostic evaluation of pancreatitis:

• It can identify abnormalities in animals with pancreatitis. In many cases, it also provides information about the severity of inflammation.

• It is noninvasive and can be repeated frequently, providing a means of following disease progression and/or resolution.

• It allows evaluation of peripancreatic structures, such as the biliary system, duodenum, and stomach, which are often secondarily involved in acute pancreatitis.

• It is an important tool for identifying complications of pancreatitis, such as biliary obstruction, abscess formation, and pseudocyst formation.

The ultrasonographic findings that characterize pancreatitis represent changes in either the pancreas itself or in peripancreatic structures. The most common ultrasonographic abnormality noted in animals with pancreatitis is a hypoechoic mass dorsomedial to the descending duodenum and caudal to the stomach. This mass represents the inflamed pancreas, and although its overall echogenicity is usually decreased, it may sometimes appear inhomogeneous.

The peripancreatic mesentery and associated fat are often hyperechoic but may have variable echogenicity. The edges of the pancreas are distinct if the inflammation is mild but become poorly defined when severe pancreatitis is present, probably as a result of the edema, necrosis, and hemorrhage that accompany severe pancreatic inflammation. The overall pancreatic image tends to become better defined with more distinct edges as inflammation subsides. Improved resolution of the pancreatic margins may be partially related to saponification of surrounding fat. The ultrasonographic changes associated with chronic pancreatitis are often less severe than those associated with acute pancreatitis, although it is difficult to differentiate one condition from another based on ultrasonography alone.

Changes in peripancreatic structures also contribute to the ultrasonographic diagnosis of pancreatitis. Free peritoneal fluid secondary to focal peritonitis may be apparent in the pancreatic region. The descending duodenum typically becomes dilated and fluid-filled, with thickened walls and no apparent peristalsis. With severe duodenitis, the duodenal wall may have a corrugated appearance.
Potential complications of pancreatitis include pseudocyst or phlegmon formation, abscess formation, and biliary obstruction. Pancreatic phlegmons are edematous masses of indurated pancreas and adjacent tissue with varying degrees of necrosis that develop within several days of the onset of acute pancreatitis. They may resolve spontaneously or may cause persistent fever and abdominal pain. Pancreatic abscesses result from secondary infection of necrotic pancreatic tissue or of a phlegmon. Ultrasonographically, pancreatic phlegmons and abscesses appear as pancreatic masses of mixed echogenicity and variable size. Gas in a pancreatic mass, identified as an echogenic interface with reverberation, suggests abscess formation.

Pancreatic pseudocysts appear ultrasonographically as primarily anechoic masses but they may contain some internal echoes. They cause mild acoustic enhancement of distal structures. Unfortunately, ultrasonography cannot usually differentiate between pancreatic phlegmons, abscesses, or pseudocysts. Extrahepatic biliary obstruction is another complication of acute pancreatitis that may necessitate surgery.

**Cytology**

Fine-needle aspiration of suspected areas of pancreatitis and suppurative inflammation may help support the diagnosis. Abdominocentesis may be helpful if effusion is present. Suppurative inflammation is the typical finding, but it is rarely septic. In addition, abdominal fluid analysis combined with measurement of abdominal fluid lipase concentration is helpful. Abdominal fluid lipase concentration higher than serum lipase concentration supports the diagnosis of pancreatitis in many cases.

**Histopathology**

Biopsy provides the definitive diagnosis. Surgery or laparoscopy is generally required to obtain a diagnostic biopsy. Pancreatic biopsy techniques are generally safe, given careful tissue handling. The primary consideration in obtaining biopsy samples is whether or not a patient with suspected acute pancreatitis is a safe anesthetic risk and whether or not the step of obtaining tissue samples from a patient will add significantly contributory information to patient management to justify any risks and also costs related to performing a biopsy procedure. Most patients that undergo pancreatic biopsy are examined during the course of a laparoscopic or surgical exploratory procedure in which various areas of the abdomen are being examined and biopsied (e.g., liver, intestines).

Although acute pancreatitis is not generally considered a surgical condition patients progressing to a more severe necrotizing pancreatitis with pancreatic abscess and peritonitis may be candidates for exploratory laparotomy.

**MEDICAL MANAGEMENT**

Initial treatment of pancreatitis should be supportive and is tailored for the individual patient, taking into consideration any abnormalities detected on physical examination and testing. Basic therapy involves correction of fluid and electrolyte imbalance, control of nausea and vomiting, pain management, nutritional considerations (early feeding is the goal), and the control of secondary complications.

Management considerations for severe and often life-threatening acute pancreatitis include pancreatic rest in the form of fasting for 1 to 3 days for vomiting patients (try to minimize the fasting period), fluid and electrolyte therapy and in some cases colloid administration, and antibiotics for severe cases or whenever there is evidence of sepsis or concurrent liver disease (antibiotics are not indicated for all cases of pancreatitis). Plasma or whole blood may be indicated in some cases. Antiemetics are given to control vomiting and decrease fluid loss and enhance patient comfort (i.e., control nausea and hopefully allow the patient to rest more and eat earlier). Maropitant given at 1 mg/kg once daily subcutaneously (SC) or intravenously (IV) (slowly over a 60- to 90-second period) is a highly effective antiemetic drug that also provides visceral analgesia, making it a very attractive antiemetic drug to use in patients that have visceral pain. Use of opioid analgesics should be strongly considered in the patient with pancreatitis even if there is no outward evidence of abdominal pain (e.g., buprenorphine for mild pain; and morphine, hydromorphone, or fentanyl for moderate to severe pain). Constant rate infusion of pure opioid drugs is an ideal way to more effectively control pain consistently hour by hour. Lidocaine infusions can also be administered when additional pain control is needed. Careful attention to detail on pain control is absolutely essential and too often inadequate therapy is administered in this area.

**Why is Early Feeding Important?**

Nutritional support of critically ill patients has long been considered a
supportive measure of low priority. Recent advances in both human and veterinary medicine have demonstrated that nutritional support is an important therapeutic modality and can aid in the management of many diseases. In diseased states, the inflammatory response triggers alterations in cytokines and hormone concentrations and shifts metabolism toward a catabolic state. With a lack of food intake, the predominant energy source is derived from accelerated proteolysis, which in itself is an energy-consuming process. Thus, critically ill animals may actually preserve fat deposits in the face of lean muscle tissue loss. The goal of nutritional support in these catabolic patients is to feed the catabolism with exogenous sources of protein and fat, thereby sparing endogenous protein which is critical to recovery.

Malnutrition in veterinary patients is thought to increase morbidity and mortality, but this has not been statistically quantified. However, nearly every body system is affected by negative energy balance. In the GI tract transit times increase, absorptive capabilities decrease, and there is an increased risk of bacterial translocation. In the kidneys, excretion of urinary calcium and phosphorus increases, ability to excrete acid decreases, gluconeogenesis increases, and glomerular filtration rate decreases. Malnutrition has been documented to decrease humoral immunity and barrier function (skin and mucosal surfaces), inflammatory response, leukocyte motility, and bactericidal activity. Patients are at risk for pulmonary complications as a result of decreased response to hypoxia, decreased lung elasticity, and secretin production, altered permeability and decreased tidal volume. Cardiovascular complications include increased incidence of arrhythmias and decreased weight of the heart muscle. Protein-calorie malnutrition may also alter the normal or expected metabolism of certain drugs, which may increase or decrease their therapeutic effect even when given at recommended dosages.

**Feeding Dogs with Pancreatitis**

Critical illnesses associated with gut barrier dysfunction include severe acute pancreatitis, inflammatory and noninflammatory bowel disease, severe burn injury, multisystem trauma, and high risk surgery. Gut barrier dysfunction can exacerbate critical illnesses by leading to bacteremia, endotoxemia, systemic inflammatory response syndrome, and multiple organ dysfunctions. The nutritional management of these disorders has traditionally included an initial period of starvation, ranging from 3 to 7 days. The most important stimulus for intestinal mucosal growth, repair, and integrity, however, is the presence of nutrients within the gut lumen. The absence of luminal nutrients leads to marked small intestinal mucosal atrophy and suppressed crypt cell proliferation, marked reductions in gut-associated lymphoid tissue cell mass and function, increased intestinal permeability to bacteria and toxins, and enhanced pro-inflammatory cytokine generation and acute-phase responses. For pancreatitis, the recommendation for starvation was based on the belief that “strict pancreatic rest” prevents stimulation of exocrine pancreatic secretion, thus protecting against autodigestion. Recently, however, this recommendation has come into question. Studies in dogs, rodents, and humans have demonstrated that exocrine pancreatic excretion is already inhibited by the inflammation associated with pancreatitis and feeding has no impact on exocrine pancreatic secretion. A systematic review of human literature found that patients with acute pancreatitis receiving enteral nutrition have fewer episodes of death, systemic infections, multiple organ failure, and operative interventions. This data suggests that enteral nutrition (EN) should be considered the standard of care for patients with acute pancreatitis requiring nutritional support.

Recent studies support the recommendation that one of the management priorities in treating acute pancreatitis should be to feed early and enterally. Prolonged fasting leads to immunosuppression, decreased wound healing and increased bacterial translocation, sepsis, and decreased survival. Ideally, canine patients should not be held NPO for more than 48 to 72 hours including the time they were anorectic prior to presentation. Once vomiting is adequately controlled, feeding should be instituted. Enteral feeding improves enterocyte health and immune function. Documented benefits of maintaining a healthy gut barrier function include:

- Reduced mucosal permeability
- Reduced incidence of bacteremia, endotoxemia, and septic morbidity
- Attenuation of the acute phase response and reduced incidence of multiple organ failures
- Reduced catabolism and preservation of a positive nitrogen balance
- And perhaps most importantly, improved clinical outcomes

Early enteral nutrition is superior to both starvation and total parenteral nutrition in critical illnesses associated with gut barrier dysfunction. One recent study comparing enteral and parenteral nutrition in dogs with acute pancreatitis documented a significantly greater number of vomiting
or regurgitation episodes in dogs receiving parenteral nutrition (PN) versus enteral nutrition. Additionally dogs receiving enteral nutrition did not demonstrate any noticeable postprandial pain. There were more catheter-related complications in the PN group. The authors concluded early EN delivered proximal to the pylorus is well tolerated in dogs with severe pancreatitis and resulted in fewer complications than PN.

Depending on the situation, enteral feeding can be done either per os or via nasoesophageal or esphagogastomy tube (see below for tube placement procedure details). Currently, prepyloric feeding via esphagogastomy tube seems to be well tolerated and this route is certainly easier than placing a jejunostomy tube. Elemental diets or polymeric diets can be fed via tube. Of the liquid diets, vanilla Ensure is specifically preferred in canine pancreatitis patients because it is lower in fat compared with other commonly used liquid diets (e.g., CliniCare, chocolate Ensure). Vanilla Ensure is not designed for long-term feeding but it is fine for the initial feeding stage in pancreatitis. For dogs with a single bout of pancreatitis, a low fat diet should be fed for the first month or two and then the patient can usually be returned to its regular diet if so desired.

Nasoesophageal Tube Placement

Nasoesophageal intubation is an easy, effective, and efficient means of providing enteral nutritional support. The availability of small bore, soft polyvinyl, and Silastic feeding tubes (i.e., 3.5 and 5 French 36 inch tubes, Argyl or National Catheter Company) and low viscosity, nutritionally complete liquid diet formulations, and patient tolerance of tube placement has made nasoesophageal tube placement a popular avenue for feeding malnourished patients. Nasoesophageal tube placement is indicated in any patient with protein-calorie malnutrition that will not undergo oral, pharyngeal, esophageal, gastric, or biliary tract surgery. Nasoesophageal tube placement is indicated in any patient with protein-calorie malnutrition that will not undergo oral, pharyngeal, esophageal, gastric, or biliary tract surgery.

**Technique:** Local nasal anesthesia, sedation, or light general anesthesia may be necessary for placement of a nasoesophageal/nasogastric tube in dogs and cats. In the majority of cases, topical anesthetic or light sedation is all that is necessary for proper tube placement.

**Anesthesia for Dogs:** Place 0.5 to 1 mL of 0.5% proparacaine hydrochloride into the nasal cavity and tilt the head upwards for several seconds to allow adequate dwell time. Repeat application of local anesthetic before attempting to pass the nasoesophageal tube. If the patient does not tolerate passage of the tube, sedation or light general anesthesia may be required.

**Tube Size:** Select an appropriate size feeding tube:

- Dogs weighing 2 to 15 kg: 5 French x 91 cm
- Dogs weighing >15 kg: 8 French x 91 cm

Estimate the length of tube to be placed in the esophagus or stomach by placing the tube from the nasal planum along the side of the patient to the seventh or eighth intercostal space (i.e., to ensure mid-esophageal placement). It is our recommendation that the feeding tube not pass through the lower esophageal sphincter, as this may result in sphincter incompetence and esophageal reflux of hydrochloric acid and other gastric content, potentially causing esophagitis. Place a tape marker on the tube once the appropriate measurement has been taken. Lubricate the tip of the tube with 5% lidocaine viscous prior to passage. Hold the patient’s head in a normal functional position (i.e., avoid hyperflexion or hyperextension).

**Confirming Esophageal Placement:** Confirm esophageal placement by injecting 3 to 5 mL of sterile saline through the tube and eliciting a cough or placing 6 to 12 mL of air and auscultating for borborygmus at the xiphoid. Placement can also be confirmed by taking a radiograph of the chest. If the patient requires general anesthesia, visually confirm tube placement in the esophagus.

**Securing the Tube to the Patient:** In the dog, the tube is secured to the lateral aspect of the nose and dorsal nasal midline with an encircling suture and Chinese finger trap or cyanoacrylate glue or both depending upon the patient’s level of activity. An Elizabethan collar should be used immediately postoperatively and until it is determined that the patient will tolerate the presence of the tube.
**Tube Management:** Place a column of water in the tube and cap it when not in use; this prevents intake of air, reflux of esophageal contents, and occlusion of the tube by diet. Three and 5 French feeding tubes come with appropriate size caps. Nasoesophageal/nasogastric tubes can be left in place for several weeks, are well tolerated, easily removed, the patient can drink and swallow around the tube, and repeated orogastric intubation is prevented.

**Esophagostomy Tube Placement**

**Indications:** Esophagostomy tube feeding is indicated in anorexic patients with disorders of the oral cavity or pharynx, or anorexic patients with a functional gastrointestinal tract distal to the esophagus.

**Contraindications:** Esophagostomy tube placement is contraindicated in patients with a primary or secondary esophageal disorder (e.g., esophageal stricture, after esophageal foreign body removal or esophageal surgery, esophagitis, megaesophagus) and patients with a history of vomiting.

**Advantages:** Advantages of esophagostomy tube feeding include ease of tube placement, tubes are well tolerated by the patient, large-bore feeding tubes can be used allowing use of blended diets, tube care and feeding is easily performed by the client, patients can eat and drink around the tube, and tube removal can be performed any time after placement. Esophageal tube placement eliminates local pharyngeal irritation, coughing, laryngospasm, or aspiration occasionally associated with pharyngostomy tubes.

**Disadvantage:** The major disadvantage of use of an esophagostomy tube is the need for general anesthesia and endotracheal intubation during placement.

**Placement Technique:** Provide general anesthesia. Place the patient in right lateral recumbency with the left side uppermost. The tube can be placed on either the right or left side of the midcervical region; however, the esophagus lies slightly left of midline making left-sided placement more desirable. Aseptically prepare the lateral midcervical area from the angle of the mandible to the thoracic inlet. Slightly extend the neck and hold the mouth open with a mouth speculum.

Premeasure and mark a 20 to 24 French feeding tube from the level of the mid-cervical region (i.e., exit point of feeding tube) to the level of the seventh or eighth intercostal space; ensuring mid- to caudal esophageal placement. Make certain the tube does not cross the lower esophageal sphincter (LES) as this may cause sphincter incompetence, gastric reflux of acid, esophagitis, and subsequent vomiting or regurgitation. Prior to tube placement, enlarge the two lateral openings of the feeding tube to encourage smoother flow of blended diets.

**Eld Esophagostomy Tube Placement Technique**

The following technique requires the use of an Eld feeding tube placement device. Place the oblique tip of the instrument shaft through the oral cavity and into the esophagus to the level of the mid cervical region (i.e., equal distance between the angle of the mandible and thoracic inlet) and palpate the tip as it bulges the cervical skin. Make a small skin incision over the device tip. Activate the spring-loaded instrument blade until it penetrates esophageal wall, cervical musculature, and subcutaneous tissue and is visible through the skin incision. Carefully enlarge the incision in the subcutaneous tissue, cervical musculature and esophageal wall with the tip of a No. 15 scalpel blade to allow penetration of the instrument shaft. Place a 2-0 Nylon suture through the side holes of the feeding tube and through the hole in the instrument blade. Tighten the suture until the tip of the instrument blade and feeding tube tip are in close apposition. Retract the instrument blade into the instrument shaft so the feeding tube tip just enters the instrument shaft (i.e., deactivating the instrument blade). Place sterile water-soluble lubricant on the tube and instrument shaft. Retract the instrument and pull the feeding tube into the oral cavity to its predetermined measurement. Remove the 2-0 Nylon suture to free the feeding tube from the instrument. Place a stylet through one of the side holes of the feeding tube and against its tip (do NOT use a stylet when placing an E-tube in cats). Lubricate the feeding tube and advance it into the esophagus until the entire oral portion of the tube disappears. Gently retract the stylet from the oral cavity being careful to ensure its release from the feeding tube. If you encounter resistance and cannot pass the feeding tube into the esophagus you may have engaged the endotracheal tube. If this happens remove the feeding tube and replace it under direct visualization. Secure the tube to the cervical skin with a Chinese finger-trap suture of No. 1 Novafil.

*A video demonstrating Eld esophagostomy tube placement can be viewed at http://www.gloydgroup.com/proceedings/videos/2014-gi-symposium-Eld-esophagostomy-tube-canine. The video is provided courtesy of Dr. Howard B. Seim III.*
Curved Carmalt Hemostat Technique

A curved Carmalt hemostat can be used to place an esophagostomy feeding tube. The curved Carmalt forceps is placed into the patient’s oral cavity with the curve of the hemostat directed toward the cervical region. The Carmalt is directed to a point equidistant between the ramus of the mandible and point of the shoulder midway between the dorsal and ventral aspect of the neck. The hemostat is pushed laterally so as to make a ‘bulge’ in the cervical region at the desired exit point described above. A scalpel blade is used to incise over the tip of the Carmalt until the tip protrudes through the skin. The tip of the feeding tube is then grasped with the Carmalt hemostat and the tube is exited out through the oral cavity. The tube is pulled out until the flanged end of the tube just comes in contact with the cervical skin. The tip of the tube is then turned back on itself, grasped with the Carmalt forceps, and redirected into the oral cavity of the dog. The tube should remain in the jaws of the Carmalt hemostat until the tip of the tube is beyond the cervical exit point of the tube. The feeding tube is then released from the Carmalt and pushed into the esophagus until the tube is in the mid-esophagus (i.e., seventh or eighth intercostal space). The tube is secured using a Chinese finger-trap friction suture.

Regardless of technique used, the exit point of the tube can be left exposed or bandaged. A column of water is placed in the tube and the exposed end capped with a 3-cc syringe; this prevents intake of air, reflux of esophageal contents, and occlusion of the tube by diet. Most patients tolerate the tube without the need of an Elizabethan collar.

Esophagostomy tubes can be removed immediately after placement or left in place for several weeks to months. Care of the tube exit site may require periodic cleansing with an antiseptic solution. Tube removal is performed by cutting the finger trap suture and gently pulling the tube. No further exit wound care is necessary; the hole seals in one or two days and heals by 7 to 10 days.

Complications: Complications associated with esophagostomy tube placement include early removal by the patient or vomiting the tube. No significant long-term complications have been reported (e.g., esophagitis, esophageal stricture, esophageal diverticulum, or subcutaneous cervical cellulitis). Reflux esophagitis can occur from improper tube placement (i.e., through the lower esophageal sphincter) or esophageal irritation from the tube itself. Mid-esophageal placement of silicone rubber tubes greatly reduces the incidence of esophageal injury and eliminates reflux esophagitis.

Other Potential Therapies

Hyperbaric oxygen therapy (HBOT) can also be very helpful in patients with severe pancreatitis (shown to be beneficial in both humans and animals). Physiological effects include:

- Oxygen delivered to the alveoli under increased atmospheric pressure results in large increases in the amount dissolved in plasma
- Increase in neovascularization
- Enhancement of white blood cell oxidative killing and antibacterial effects
- Inhibition of neutrophil adherence to microvascular endothelia resulting in suppression of the deleterious cascade of events that follows in ischemia-reperfusion injury
- Modification of cytokine effects (anti-inflammatory)
- Inhibition of free radical formation
- Down regulation of intercellular adhesion molecule (ICAM-1) expression

A 2012 review article on the pathophysiology of pancreatitis by Caroline Mansfield in the Journal of Veterinary Internal Medicine implicates perpetuation of inflammation in pancreatitis by the adhesion of leukocytes to endothelial walls via expression of ICAM-1 and selectins mediated by IL-8. In addition, a disturbance in pancreatic microcirculation with ensuing ischemia is implicated as a major factor in the ongoing cycle of inflammation associated with pancreatitis. For veterinarians who have access on a referral basis to a hospital with a hyperbaric chamber HBOT is an exciting modality that can be used in addition to all of the other high priority therapeutic modalities already discussed.

Unproven therapy should be considered only after careful evaluation of the individual case and may include corticosteroids; somatostatin, a hormone that decreases pancreatic secretion; and low dose dopamine, which was found to preserve vascular permeability during experimental pancreatitis in cats and may be a beneficial adjunctive therapy in the management of clinical pancreatitis. Antioxidants may be of benefit in the acute management of pancreatitis; vitamin E is a potent membrane antioxidant and SAMe (S-adenosyl methionine) replaces glutathione stores that may have some benefit in pancreatitis, peritoneal lavage removes inflammatory products in the peritoneal cavity before they are absorbed, and pancreatic enzyme supplementation has been reported to decrease the pain that
accompanies chronic pancreatitis in humans by the feedback inhibition by endogenous pancreatic enzyme secretion. It is not known if enzymes are helpful in the acute cases but such supplementation may have some benefit in early nutrition of patients with acute pancreatitis.

**SURGICAL MANAGEMENT OF PANCREATITIS**

Surgery is rarely indicated for pancreatitis; however, there are situations in which surgery may be necessary. Indications may include pancreatic abscess, septic suppurative peritonitis secondary to severe necrotizing pancreatitis, pancreatic pseudocyst, jejunostomy feeding tube placement, and open peritoneal lavage and drainage. Surgery of the pancreas is discussed in current surgery textbooks.

**CONCLUSION**

Acute pancreatitis can vary in severity of signs and often results in multiple organ system involvement. Despite extensive literature on pathogenesis of pancreatitis and its complications, there have been few notable advances made in its medical and surgical management. It is possible that future research on modification of enzymatic disturbances will result in an effective treatment for acute pancreatitis. Surgical treatment of pancreatitis remains a controversial topic and is generally reserved for patients with severe necrotizing pancreatitis with septic peritonitis or pancreatitis associated with pancreatic mass.

**REFERENCES**

VIDEO – Eld Esophagostomy Tube Placement
Gastric Antral Mucosal Hypertrophy

David C. Twedt, DVM, DACVIM • David S. Biller, DVM, DACVR • Howard B. Seim III, DVM, DACVS

SUMMARY

Diagnosing chronic vomiting caused by pyloric outflow disorders requires a systematic approach excluding other causes of vomiting. Specific imaging techniques, including ultrasound and contrast studies, and endoscopy findings can confirm the diagnosis. A syndrome of gastric antral-pyloric mucosal hypertrophy is a common and treatable cause of pyloric disorder in the dog. Surgical management is required; the prognosis is very good following the most commonly performed pyloroplasty procedures, pylorocentesis and Y-U pyloroplasty.

CRITICAL KEYS

- Vomiting of undigested food up to 6 to 8 hours after eating suggests a gastric motility or obstructive disorder.
- When vomiting due to abnormal gastric retention of food occurs obstructive disease must first be ruled out. If there is no obstruction, gastric hypomotility is more likely the cause.
- Ultrasound evaluation of the stomach may reveal gastric wall thickening that can be confirmed with barium contrast studies.
- Endoscopy is a means of evaluating the gastric lumen for masses or ulcerative lesions and to enable mucosal biopsies.
- Gastric antral mucosal hypertrophy (GAMH) is characterized by large redundant rugal folds that can be seen on endoscopy. The folds “feel” normal when palpated with biopsy forceps, in contrast to the folds that may be seen with adenocarcinoma or lymphoma, which have a similar appearance but are generally hard and firm.
- Feeding small liquefied meals may alleviate signs in mild cases but severe cases will require a pyloric mucosal resection with a pyloric-opening surgery.
- Surgery of antral-pyloric mucosal hypertrophy is successful in the majority of cases. Pyloroplasty may be successful in mild cases while severe cases require pylorocentesis.
- The prognosis for GAMH is very good following successful surgical correction. A reported 80% or higher response rate is expected.

Gastric antral mucosal hypertrophy (GAMH) is an idiopathic condition characterized by excessive hypertrophy of the mucosa in the antral region of the stomach. The large mucosal folds cause obstruction of gastric outflow. Some of these folds may appear as mucosal polyps on endoscopy. The cause of mucosal hypertrophy is unknown. Trophic factors, such as gastrin, histamine, and increased innervation, have been suggested but are only theories. Infectious causes such as Helicobacter are not thought to be involved. This condition is seen predominantly in older small-breed dogs. Clinical signs are generally associated with vomiting suggestive of an outflow obstruction. Generally the stomach should be empty of a meal in 6 to 8 hours and vomiting of undigested food more than 8 hours after a meal suggests either a motility or outflow obstructive disorder. Most affected
dogs are healthy and have a good appetite but have chronic intermittent vomiting. Weight loss can occur in some cases. Rarely, complete gastric outflow obstruction can result in dehydration with a metabolic alkalosis.

Imaging studies (see below) suggest obstruction and ultrasound may demonstrate a mucosal thickening. Contrast radiology will demonstrate an antral lesion. Endoscopy helps confirm the diagnosis. In the normal dog the antrum is free of rugal folds; in affected dogs the antral mucosa contains large redundant rugal folds that can be seen on endoscopy. These changes can also occur with neoplasia (adenocarcinoma or lymphoma); however, in neoplasia these folds are generally hard and firm. With mucosal hypertrophy the mucosa is not firm and “feels” normal when palpated with the biopsy forceps. The biopsies of dogs with GAMH generally reveal normal mucosa with variable inflammatory infiltrates with or without *Helicobacter*. The pyloric canal is normal and usually one can easily pass the endoscope into the duodenum.

Feeding small liquefied meals may alleviate signs in mild cases but severe cases will require a pyloric mucosal resection with a pyloric opening surgery as is discussed below.

**IMAGING STUDIES**

**Normal**

The radiographic anatomy of the stomach is variable and dependent upon species, breed, conformation, degree of gastric distention, volume and type of gastric contents, and position of the patient during the exposure. The stomach is usually within the rib cage. On the lateral radiograph the vertical axis of the stomach should be approximately parallel to the tenth or eleventh intercostal space. In deep-chested dogs the gastric axis may be perpendicular to the spine. On the ventrodorsal radiograph in the dog the cardia, fundus, and body are located to the left of midline. The pyloric portions are to the right of midline. Rugal folds are not well visualized on survey radiographs. If there is negative (gas) or positive contrast (iohexol or barium) rugal folds may be seen. The size and number of rugal folds will vary depending on the degree of gastric distention; therefore, assessment is very subjective.

Standard survey abdominal radiographs include a ventrodorsal and either a left or right lateral recumbent view. The determination of which lateral radiograph is obtained is often personal preference. There are differences in the appearance of various organs, such as the kidneys, spleen, and gastrointestinal tract, between the right and left lateral recumbent radiograph. This varied appearance is particularly noticeable in the gastrointestinal tract, which is dependent on gas to provide contrast for visualization of the mucosal surface. Fluid and gastric contents are extremely mobile and tend to move to the dependent portion of the stomach during postural changes. Gas will rise to the non-dependent portion of the stomach. For example, in right lateral recumbency gas accumulates in the fundus, and in left lateral recumbency redistribution to the pyloric region occurs.

Gas within the gastrointestinal tract serves as a negative contrast medium. Specifically, the change in positioning of the animal for the opposite lateral abdominal radiographs will allow for the redistribution of gas already present in the stomach. It is important to realize that the amount of gas will have an effect on which portions of the stomach will contain gas. In a severely gas-distended stomach, gas may be in all portions of the stomach on both lateral abdominal radiographs. Even in these situations the location of the pylorus can be determined. A fluid-filled pylorus is an area that can be misdiagnosed as a cranial abdominal mass when the right lateral recumbent radiograph is taken. When the left lateral abdominal radiograph is obtained the pylorus will be filled with gas.

Delay in gastric emptying may be secondary to a mechanical or functional problem. Pyloric obstructions may be due to hypertrophic changes, foreign bodies, and neoplasia. A primary rule-out for what radiographically appears as a moderately distended stomach and suspicion of pyloric obstruction is motility or functional problems due secondarily to drugs such as atropine, antispasmodics, or certain tranquilizers.

**CONTRAST STUDIES**

With outflow obstruction, gastric emptying time may be delayed from 3 to as long as 6 hours. With contrast material, the pyloric canal may appear
narrowed. Contrast may be seen extending through this concentrically narrowed and elongated pylorus and identified as a “beak” (bird’s beak) or “string” sign. The pyloric “tit” is the peristaltic pouch, or out-pouching of the pyloric antrum along the lesser curvature, as a peristaltic wave pushes contrast medium against the outflow obstruction.

ULTRASOUND

Ultrasound has been applied successfully to the diagnosis of a number of gastrointestinal disorders, including gastric foreign bodies, uremic gastropathy, chronic pyloric hypertrophic gastropathy, and gastric neoplasia. Ultrasound has proven useful not only in the diagnosis of morphologic disease but also in the evaluation of gastric function. Maximizing resolution by using a high frequency transducer is critical in the examination of the stomach. Fasting the animal prior to ultrasonography also improves the results of ultrasound examination. A high frequency transducer is important (7.5–14 MHz) to maximize resolution. As with all ultrasound studies, a complete exam of the entire abdomen should be performed to ensure that concurrent disease is not missed. For example, GI disease is often associated with mesenteric lymphadenopathy or secondary to pancreatic disease.

Complete ultrasonographic examination of the stomach includes evaluation of wall thickness and layering, evaluation of luminal contents, and quantifying peristaltic function. The stomach should be scanned in both the longitudinal and transverse planes. As with radiographs the appearance will vary depending on the degree of distention and the luminal contents. In the normal dog the gastric wall is 3 to 6 mm thick when the stomach is moderately distended and may be slightly thicker when the stomach is not distended. These thickness measurements of the stomach are taken between rugal folds. Gastric rugae can be recognized in the fundus and body of the stomach, and their thickness depends on the degree of gastric distention.

Ultrasonography also allows for differentiation of the layers of the stomach, which alternate in echogenicity. Under optimal conditions, five separate layers can be identified:

- Luminal–mucosal interface (hyperechoic)
- Mucosa (hypoechoic)
- Submucosa (hyperechoic)
- Muscularis (hypoechoic)
- Subserosa-serosa (hyperechoic)

The submucosa and subserosa-serosa are hyperechoic because of the presence of relatively more fibrous connective tissue. The mean number of peristaltic contractions in the stomach is four to five per minute.

The ultrasonographic appearance of the stomach lumen depends on its contents. In a collapsed state the stomach lumen appears as a hyperechoic core (“mucosal stripe”) surrounded by a hypoechoic halo. This hyperechoic core represents mucus and small air bubbles trapped at the mucosal–luminal interface. When fluid is present in the lumen, an anechoic area is present between the walls. Gas within the stomach lumen causes a highly echogenic interface with a reverberation artifact. The presence of fluid in the lumen improves the sonographer’s ability to evaluate the mucosal and submucosal layers, whereas the presence of luminal gas hinders it.

Ultrasonographic findings characteristic of chronic antral mucosal hypertrophy include gastric distention and thickening of the pyloric wall. Examination of the pylorus in a transverse plane shows an evenly thick hypoechoic ring (representing the muscularis) surrounding the pyloric lumen. The thickness of the pyloric wall can be greater than 9 mm and the thickness of the muscular layer can be greater than 4 mm.

SURGERY

The major indication for pyloric surgery is gastric outlet obstruction (i.e., pyloric stenosis, pyloric polyps, GAMH, antral neoplasia). The most commonly performed pyloroplasty procedures are pyloroplasty and Y-U pyloroplasty. Rarely are procedures such as pyloromyotomy or transverse pyloroplasty indicated.

Pyloromyotomy

This procedure is used for patients with pyloric stenosis secondary to pyloric muscular hypertrophy. The technique involves cutting the muscular layers of the pylorus down to the level of the mucosa until the mucosa bulges. Pyloric muscular hypertrophy rarely occurs in dogs and cats.

Transverse Pyloroplasty

This procedure is used in patients with pyloric obstruction secondary to mild pyloric mucosal hypertrophy or pyloric polyps. The technique involves a full thickness longitudinal incision through the pylorus and suturing the incision transversely. Closure is performed with a single layer of simple interrupted synthetic absorbable sutures placed through all layers of the bowel wall.
**Y-U Pyloroplasty**

This procedure is performed when a transverse pyloroplasty will not provide a large enough opening in the obstructed pylorus, and when more exposure of the pylorus is needed to resect redundant mucosa. An incision is made from the gastric side of the pylorus, through the pylorus and into the duodenum approximately 2 to 3 cm long (depending upon breed size). The second and third incisions are made in the antral portion of the stomach forming the limbs of the Y. Each limb of the Y incision should be of equal length (2–3 cm). The apex of the Y incision is sutured to the apex of the duodenal incision. The newly opposed edges for a U-shaped configuration and are sutured together using a simple continuous or simple interrupted appositional suture pattern with synthetic absorbable monofilament suture material.

**Pylorectomy**

This procedure is most commonly performed in patients with GAMH in which the pathologic process involves all layers of the stomach and pylorus. In these severely affected patients, complete removal of the pylorus is necessary to allow proper gastric emptying to resume. The pylorus is carefully palpated to determine the margins of pylorus affected. Doyen intestinal forceps are used to occlude the gastric antrum and duodenum 3 to 4 cm beyond the proposed sites of pyloric resection. Straight Metzenbaum scissors are used to excise the pylorus. The remaining lumens to be sutured are the gastric antrum (large diameter) to the duodenum (small diameter). The gastric antrum is sutured together using a simple continuous appositional pattern until its lumen diameter approaches the same size as the duodenal lumen diameter. A stay suture is then placed in the mesenteric border of the gastric and duodenal segments and used to gently bring the two segments into apposition. Gastric and duodenal lumen diameters are examined to ensure they are exactly the same. A second stay suture is placed in the antimesenteric border of the antral and duodenal segments bringing the two into perfect apposition. Traction and counter-traction is placed on each stay suture thus bringing the two lumens into apposition. Suturing is commenced with a simple continuous appositional suture pattern using a synthetic absorbable suture. Once the anastomosis is complete the stay sutures are removed, the Doyen intestinal forceps removed, and the suture line is leak checked. Intraoperative placement of a jejunostomy feeding tube should be considered in patients that undergo gastroenterostomy procedures.

*A video demonstrating pylorectomy can be viewed at http://www.gloydgroup.com/proceedings/videos/2014-gi-symposium-pyrorectomy-GAMH-canine. The video is provided courtesy of Dr. Howard B. Seim III.*

**PROGNOSIS**

The prognosis for GAMH is very good following successful surgical correction. A reported 80% or higher response rate is expected. It is always important to perform histopathology on the resected tissue to assure that infiltrative disease is not present. Small frequent semi-liquid meals may be required in some cases. In rare situations gastric prokinetic agents may be required to improve emptying. It is possible that long-standing gastric distention may lead to secondary gastric hypomotility.

**REFERENCES AND SUGGESTED READING**

Inflammatory Bowel Disease and Intestinal Lymphoma in Cats

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SUMMARY

Inflammatory bowel disease (IBD) and lymphoma of the small intestine are common causes of GI disease in middle-aged to old cats. It is important that an accurate diagnosis is made so that the correct therapy is provided, quality of life is enhanced, and lifespan is maximized. This session will examine the challenge of diagnosis, why taking the steps to make a diagnosis is far superior to trying empirical therapy, the pros and cons of endoscopic versus surgical biopsies, and the latest recommendations for management of IBD and GI lymphoma.

CRITICAL KEYS

- A thorough diagnostic approach is necessary for patients with weight loss as there are many potential causes.
- Cats with an increased appetite and weight loss should be investigated for hyperthyroidism. Another cause that should always be considered in older cats, although not a common disorder, is exocrine pancreatic insufficiency (EPI). Not all cats with EPI have diarrhea.
- Cystic masses in the liver are fairly common in older cats and are usually not a cause of any significant clinical signs—i.e., they are often an incidental finding. Caution is in order as other more important problems may be missed if the diagnostic approach is not thorough, looking beyond the presence of the cystic liver mass.
- Ultrasonography can provide valuable diagnostic information in most cats with an abdominal mass if it is done properly and with patience by an experienced sonographer.
- There are two main types of gastrointestinal (GI) lymphosarcoma in cats: an acute lymphoblastic form and a chronic low grade lymphocytic form. Cats with the low grade form can have clinical signs identical to those in cats with inflammatory bowel disease (IBD).
- GI biopsies are essential for differentiating IBD from lymphoma. Biopsies can be obtained either through upper and lower GI endoscopy or at exploratory laparotomy. The ileum should always be biopsied in cats in addition to obtaining biopsies from the upper small intestine.
- With appropriate therapy, cats with chronic low grade lymphocytic lymphoma can live for many months to years.
**INFLAMMATORY BOWEL DISEASE**

Inflammatory bowel disease (IBD) is not a specific diagnosis; rather, it is a histologic description of a syndrome resulting from a host hypersensitivity response to antigenic stimuli. In IBD there is an increase in the inflammatory cell population in the intestinal mucosa. The predominant inflammatory component in cats with IBD can be lymphocytic-plasmacytic (most common type), eosinophilic, or neutrophilic. Changes in mucosal architecture and cell morphology should also be noted (crypt lesions including abscesses, villus atrophy or fusion, edema, epithelial erosions or ulceration, fibrosis). The etiology of IBD is poorly understood. Primary causes of initiation and perpetuation of intestinal inflammation that should be considered include parasites, bacteria (specific agents including normal luminal bacteria or bacterial overgrowth), immune-mediated diseases, and food sensitivities. Many cases of IBD are likely idiopathic in nature.

**Clinical Course**

Inflammatory bowel disease currently is recognized as a common and important medical problem in cats. Three general types of clinical presentations have been identified in cats with idiopathic IBD:

- a clinical course characterized primarily by vomiting
- a clinical course characterized primarily by diarrhea
- a clinical course that includes both vomiting and diarrhea as primary signs

Associated clinical signs can include change in appetite (anorexia, inappetence, or ravenousness), weight loss, and lethargy. In some cats, the clinical signs are cyclic; they seem to flare up and then abate in a predictable pattern.

Vomiting, one of the most frequent clinical signs of IBD in cats, is most often recognized as an intermittent occurrence for weeks, months, or years. As the disorder progresses, an increased frequency of vomiting often leads the cat’s owner to seek veterinary attention.

In addition to vomiting, diarrhea is a common sign observed in feline IBD and most likely is due to derangement of normal mechanisms of absorption and motility caused by mucosal inflammation. In most cases, diarrhea is intermittent early in the course of the disorder, and there may be a transient response (weeks to several months) to dietary manipulation or any of a variety of medications (in some cases, however, dietary manipulation can effect excellent control and drug therapy may ultimately not be necessary). Later, the diarrhea becomes persistent and usually responds only to specific treatment, which is determined after a definitive diagnosis is made. Signs of small bowel diarrhea predominate, but signs of large bowel diarrhea may be evident as well if there is generalized intestinal tract involvement.

Appetite changes in cats with idiopathic IBD vary from decreased appetite to complete anorexia to ravenousness. Inappetence seems to occur more commonly in cats that have vomiting as the primary clinical sign and usually occurs during exacerbation of clinical signs, and vomiting or diarrhea is not observed until later or not at all. The three leading differential diagnoses for a cat with a ravenous appetite, diarrhea, and weight loss are IBD, hyperthyroidism, and exocrine pancreatic insufficiency (uncommon).

**Diagnosis**

A definitive diagnosis of IBD can be made based only on intestinal biopsy performed either at endoscopy or exploratory laparotomy, and ensuring that both upper and lower (ileum) biopsies are obtained. A definitive diagnosis of IBD cannot be made based on barium series radiography or ultrasonography. Diagnostic workup prior to performing biopsies includes baseline testing to evaluate the overall health status of the patient and to rule out other disorders. Recommended baseline tests include a complete blood count, complete biochemical profile, urinalysis, fecal exams for parasites, serum thyroxine (T4) test, serum cobalamin level, and feline leukemia virus/feline immunodeficiency virus (FeLV/FIV) testing. Cats with chronic vomiting should be screened for heartworm disease. Feline trypsin-like immunoreactivity (fTLI) is done to rule out exocrine pancreatic insufficiency. Ultrasonography is useful for assessing the abdominal organs, intestinal wall thickness, searching for any masses, and examining for lymphadenopathy. Dietary sensitivity is a common problem in cats with vomiting and/or diarrhea and food trials are an important part of the diagnostic workup, especially early in the clinical course. Hydrolyzed protein and novel protein foods should be fed for 2 to 3 weeks at a time to determine if dietary therapy will either reduce or resolve the problem entirely.
ABDOMINAL IMAGING IN CATS – IBD VERSUS LYMPHOMA

Radiology

Radiography is important for diagnosing intestinal diseases. During evaluation of the small bowel on survey radiographs, important factors that should be evaluated include:

- Location of small intestine (normally fills the abdomen where nothing else is present, not unusual to be mostly right-sided in cats)
- Appearance of bowel contents (gas, fluid, or mottled material)
- Contour of small bowel
- Diameter of the small intestine (normal diameter in cats is up to 12 mm)

In normal animals, intestinal luminal contents should appear as a homogeneous fluid opacity. Disease of the small intestine may be missed on survey films unless there is a change in bowel opacity (mineralized mass or foreign material), luminal diameter (functional ileus or complete or partial mechanical obstruction), or changes in contour of the small bowel (linear foreign body).

Contrast studies (upper GI series) are often necessary to identify normal or abnormal shape, diameter, or continuity of small bowel. The transit time of barium varies greatly in cats. It usually travels from the stomach through to the ileum in about 60 minutes, although it can take as long as 4 hours. The range of transit times for organic iodides through the small bowel is approximately 15 to 90 minutes. The organic iodide usually reaches the ileum and colon in less than 60 minutes.

Small Intestinal IBD

Diagnostic radiographs are recommended in the workup of cats with GI signs. Although survey and contrast radiographs are usually not specific/diagnostic for IBD, abdominal radiography is most helpful in defining extra-alimentary tract disorders causing gastroenteritis. Survey radiography might detect organomegaly (liver, kidney) unrelated to IBD or intestinal obstruction that might cause similar GI signs. Survey radiographs of IBD are usually normal. There is no consistent radiologic finding in cats with IBD. The intestines may appear thickened (intestinal thickness cannot adequately be determined on survey radiographs), or luminal fluid maybe increased and there may be more gas than normal in the intestines, but these signs can occur in many conditions. Contrast examinations (upper GI series) are helpful in identifying a mass or obstruction. With contrast, assessment of the location and extent of the intestinal lesion may be more accurate than on survey images. Changes associated with IBD on barium study are often not present. With severe inflammation, however, changes may include irregular mucosal lining abnormalities and thickened intestinal walls. In most cases contrast radiography is unrewarding.

Intestinal Lymphoma

Survey radiography might detect organomegaly (liver, kidney, lymph nodes) associated with lymphoma. Radiographic findings may reveal a mid-abdominal mass associated with the GI tract and/or mesentery, or localized or diffuse decrease, or loss, of serosal detail suggestive of peritoneal effusion. If a mass is suspected radiographically or historically, or a mass has been palpated, then compression radiography may be helpful in isolating and visualizing the mass. Obstruction occurs more often with adenocarcinoma of the small intestine than with small intestinal lymphoma. Contrast examinations (upper GI series) are helpful in identifying the mass or the obstruction. With contrast, the location, bowel wall thickening, mucosal irregularity, and extent of the intestinal lesion may be more accurate than on survey images.

Ultrasonography of the Feline Small Intestines

The small intestines can be seen throughout the abdomen, both end-on and longitudinally oriented. The duodenum has a slightly larger diameter than the rest of the small intestinal loops, and is the most lateral and ventral bowel loop in the right cranial abdomen. It can be located usually just ventral and lateral to the right kidney and followed cranially into the pylorus. The ileum has a distinct cross-sectional appearance (resembling spokes on a wheel) and can be visualized as it enters the colon, just medial to the right kidney. The colon typically is gas-filled, with poor visualization of the lumen.

The following five layers are present in the intestinal wall, from outside to inside:

- Serosa: Thin hyperechoic layer
- Muscularis: Thin hypoechoic layer
- Submucosa: Thin hyperechoic layer
- Mucosa: Prominent hypoechoic layer (typically the thickest layer)
- Mucosal surface-lumen interface: Hyperechoic layer in the center of the bowel
These individual layers are best visualized with higher-frequency transducers.

Normal wall thicknesses have been established in the cat for various segments of the GI tract:

- Duodenum: 2.0–2.4 mm (mean 2.2 mm)
- Jejunum: 2.1–2.5 mm (mean 2.3 mm)
- Ileum: 2.5–3.2 mm (mean 2.8 mm)
- Colon: 1.4–1.7 mm (mean 1.5 mm)

One to three contractions per minute should be seen with normal small intestinal peristaltic activity.

Ultrasonographic features of intestinal disease include bowel wall thickening, loss of wall layers, loss of motility, and regional lymph node involvement.

**Intestinal Ultrasound: IBD Versus Lymphoma**

An abdominal ultrasound examination may be helpful in cases of suspected small intestinal disease. Abdominal ultrasound is superior to radiology in defining focal versus diffuse disease, loss of layering, intestinal thickening, and mesenteric lymphadenopathy seen with IBD and lymphoma. Ultrasonography also allows for precise guidance of fine needle aspiration or biopsy for cytologic or histopathologic sampling of small intestinal disease and associated lymphadenopathy.

Ultrasonography can also be used to assess response to therapy noninvasively. A limitation of ultrasonography would be the difficulty in assessing the exact anatomic location (duodenum and ilium should be more easily identified by an experienced operator). Findings may be normal, especially in cases of low-grade small cell lymphoma or mild IBD. Changes of the small intestine may or may not be present dependent upon chronicity and/or severity. The changes may be diffuse or focal. The intestine may appear normal. Biopsy is indicated to confirm disease.

The most common finding with inflammation is normal to symmetric wall thickening with the layering retained. In comparison, neoplasia is usually localized with greater wall thickness and loss of normal layering. These categories can overlap, and therefore cytology or histopathology is required for definitive diagnosis. Acute enteritis or IBD may demonstrate corrugation of the intestine on ultrasound examination.

### Ultrasound of IBD

With inflammatory bowel disease, the intestine may be normal on ultrasound. The measurement of the intestinal wall thickness by ultrasound is neither specific nor sensitive for diagnosing IBD. Changes, especially those of severe or chronic disease, have been reported as focal to diffuse thickening, altered echogenicity, poor intestinal wall layer definition, and mild enlargement of adjacent lymph nodes. Mucosal echogenicity may remain hypoechoic. Round, enlarged, hypoechoic lymph nodes may be more consistent with neoplasia, while inflammatory lymph nodes may be enlarged but tend to maintain their normal shape (Table 1).

<table>
<thead>
<tr>
<th>US Length (mm)</th>
<th>US Diameter (mm)</th>
<th>Frequency of Detection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jejunal</td>
<td>20.2 (11.4–39.0)</td>
<td>5.0 (2.8–7.2)</td>
</tr>
<tr>
<td>Colic</td>
<td>9.0 (4.6–12.1)</td>
<td>3.1 (1.9–5.2)</td>
</tr>
</tbody>
</table>

Table 1. Ultrasonographic Measurements of Feline Abdominal Lymph Nodes

### Ultrasound of Intestinal Lymphoma

Perform abdominal ultrasonography to evaluate the extra-intestinal organs in addition to GI tract wall thickness, layering, and motility. Lymphoma most commonly presents as transmural, circumferential, homogeneous, hypoechoic thickening with loss of normal wall layering. Lymphoma tends to involve a long bowel segment or multiple bowel segments. Regional moderate, hypoechoic lymphadenopathy is generally present. Lymphoma is less likely to cause obstruction of the lumen.

Six major patterns of ultrasonographic features in feline lymphoma include:

- Transmural – circumferential
- Symmetrical and asymmetrical
- Transmural – bulky
- Transmural – nodular
- Transmural – segmental
- Mucosal infiltration

The transmural–circumferential pattern is most common. The transmural–bulky pattern has been described as a space occupying mass representing the thickened wall with areas of increased and decreased echogenicity.
The transmural–segmental pattern has been described as wall thickening involving only a portion of the wall. The transmural–nodular pattern appeared as nodular wall infiltration and local nodular spread into the mesentery. Mucosal infiltration pattern demonstrated mild thickening of the intestinal wall associated with faint hyperechoic foci throughout thickened mucosal layer. In cats GI lymphoma can affect the intestinal tract without disrupting the wall layering.

Ultrasonographic Evaluation of Muscularis Propria in Cats with Diffuse Small Intestinal Lymphoma or IBD

It is difficult to detect small intestinal lymphoma or IBD in cats without a mass lesion, loss of layering or thickened bowel wall. Thickening of the muscularis propria is associated with diffuse infiltrative bowel disease such as lymphoma or IBD in cats. This has also been seen in normal cats as well. The most common ultrasound descriptions of GI lymphoma in cats are as mass lesions previously discussed.

INTESTINAL BIOPSY TECHNIQUES

Endoscopic Biopsy

Endoscopy is a minimally invasive procedure in which multiple biopsies can be obtained; this procedure generally is more appealing to clients because it is less invasive and less expensive than exploratory abdominal surgical procedures. Endoscopy is considered a gold standard procedure for tissue collection. Operator experience and the quality and number of biopsy samples obtained are very important. Endoscopy offers a means of examining the upper and lower small intestine, stomach, and colon. It is especially advantageous because biopsies can be obtained early in the course of the disorder, at a stage when a client will likely be reluctant to agree to an exploratory surgery for their pet. The degree of intestinal changes noted on biopsy also provides useful guidelines for both type and duration of therapy that will be needed to control the specific disorder.

Clinicians need to make sure they are taking an adequate number of endoscopic biopsy samples for accurate diagnosis. Even expert endoscopists report that in some cases one fourth to one third of the biopsy samples they take from a patient will have some degree of damage to the tissue that may preclude the samples from being useful or representative. Therefore, it is recommended that clinicians take 8 to 12 biopsy samples from the upper small intestine so that the pathologist will have enough tissue to work with. Also, it is recommended that both upper and lower GI endoscopy be done on cats with chronic GI signs (vomiting and/or diarrhea, weight loss). In this way biopsies from the ileum can be obtained by passing the endoscope along the full length of the colon up to the level of the ileocolic orifice. **It is very important that the effort be made to obtain ileum samples because some cats with small cell lymphoma have disease in the ileum but not in the upper small intestine.** The diagnosis can be missed in these cats if only upper small intestinal biopsies are obtained.

When a pediatric-diameter endoscope is used it is possible in most dogs over 4 to 5 kg to advance the endoscope through the ileocolic orifice and into the ileum, where it can then be advanced along the terminal ileum for exam and biopsies. In cats, however, the ileocolic orifice is very small and in most cats it is not possible to advance the endoscope through this junction and into the ileum. In cats ileum biopsies are obtained blindly by advancing the endoscopic biopsy instrument through the ileocolic orifice with the endoscope tip positioned at the ileocolic sphincter area. Usually three to four samples are procured in this way. Colon biopsies are always obtained as well during colonoscopy in order to evaluate for inflammation in the colon.

Surgical Biopsy Techniques for Abdominal Organs

Organ biopsy is usually required to confirm feline IBD and lymphoma. This can be accomplished using either laparoscopic techniques or open abdominal surgery. Laparoscopic techniques have been well described for organ biopsy. These techniques are minimally invasive and well suited for tissue procurement; however, laparoscopy is not yet readily available as a diagnostic tool in most small animal clinics. Surgery, on the other hand, is an excellent way to obtain liver, pancreatic, and intestinal biopsies. In addition to biopsy the liver should be cultured as well as bile aspirates for culture and cytology. We also currently culture the pancreas as well during laparotomy.

Intestinal Biopsy*

Several techniques can be used to successfully biopsy the intestine. Always remember: FULL THICKNESS biopsy is mandatory for the pathologist to give you the most accurate diagnosis.

When taking an intestinal biopsy, the easiest way to guarantee you will get an adequate size, full-thickness piece of intestine is to use a brand
new 4-mm skin punch biopsy instrument. The skin punch is placed on the antimesenteric border of the proposed segment of intestine and ‘drilled’ through all layers of intestine until the biopsy punch can be felt to enter the lumen of the intestine. The skin punch is removed and the biopsy retrieved from the shaft of the skin punch biopsy. This technique is particularly useful for ileal biopsy as it is easy to biopsy between the mesenteric and antimesenteric vessels. Transverse closure of the biopsy site is recommended to eliminate the possibility of lumen compromise. Suture technique is as described above for enterotomy closure. This is the author’s (H.S.) preferred technique for intestinal biopsy.

An alternate technique for intestinal biopsy is to make a 2- to 3-mm-long incision on the antimesenteric border of the intestinal segment. A No. 11 or No. 15 BP scalpel blade is used to penetrate the intestinal wall. The blade is withdrawn to create a 2- to 3-mm-long incision. A second parallel incision is made 1 to 2 mm from the original incision. A DeBakey forceps is used to grasp one end of the parallel incisions; a Metzenbaum scissor is used to cut out the piece of intestine. The surgeon should be careful not to crush the specimen with forceps. Only handle one end of the specimen while excising the biopsy specimen. If excessive trauma is created during biopsy, the pathologist may not be able to determine if the pathology is real or surgically created. The excised piece of intestine is examined closely to ensure that all layers have been included in the specimen. The biopsy site is closed using a simple interrupted or simple continuous suture pattern. 3-0 or 4-0 monofilament absorbable suture with a swaged-on sharp taper or taper-cut (penetrating point) needle is recommended. Care is taken to ensure that at least 3-mm bites are taken into the intestine and the sutures are no more that 2 to 3 mm apart.

Biopsy of the duodenum, jejunum, and ileum is recommended whenever a chronic vomiting/diarrhea patient is explored.

Complications associated with multiple intestinal biopsies are rare, even in patients that present with protein-losing enteropathy. One study looking at the complication rate of intestinal surgical procedures in patients with normal protein levels and patients that were hypoproteineemic found no difference. Complications in patients undergoing intestinal surgical procedures are generally related to the surgeon’s technical ability, not the patient’s preoperative status.

**Lymph Node Biopsy**

All lymph nodes are encased in a layer of peritoneum. When performing a lymph node biopsy it is best to tent the peritoneal covering with forceps and incise it with metzenbaum scissors. The peritoneum is then gently dissected off the lymph node. The exposed lymph node is biopsied using a #15 or #11 scalpel blade. Generally, a thin section of lymph node is ‘filleted’ off and placed in a moistened gauze sponge. The peritoneum covering the remaining lymph node is sutured to create suture pressure to help control surface hemorrhage.

**Liver Biopsy**

Surgical biopsy techniques during exploratory laparotomy are described here. The simplest method is performed by cutting a strip of liver parenchyma 5 to 6 mm thick along the border of the liver lobe. Excessive bleeding is rarely a problem with this technique; hemorrhage is controlled via cautery or direct pressure. Diffuse liver disease must be present if this method is to be diagnostic.

A second technique involves placing an encircling ligature around a pedicle of liver tissue. As the ligature is tightened, it cuts through the hepatic parenchyma, ligating hepatic vessels and bile ducts. This technique, widely known as the Guillotine technique, has been criticized for leaving excessive amounts of devitalized parenchyma. This can be avoided by inserting scissors through the cut parenchyma and cutting hepatic vessels and bile ducts just distal to the ligature. This method requires the presence of diffuse liver disease to obtain a diagnostic biopsy unless the lesion is present in the distal aspect of the liver lobe.

More localized abnormalities can be biopsied by wedge resections or partial lobectomy. Wedge resections may be performed by placing a row of overlapping, full-thickness, interrupted mattress sutures of 0 or 2-0 Maxon or Biosyn along each side of the wedge to be removed; these sutures should commence at the edge of the liver lobe and meet proximally to form a “V.” The sutures should be tied so as to compress the liver slightly but not cut into liver parenchyma. The wedge of tissue to be removed is incised about 5 mm from the suture line. Alternatively, the wedge may be removed prior to tightening the mattress sutures; preplaced mattress sutures are then gently tied with enough tension to control bleeding.

An alternate technique for use in patients with diffuse fibrotic liver disorders is performed by penetrating the affected liver lobe with a straight mosquito hemostat. The hemostat tip is placed on the surface of the liver lobe to be biopsied and gently plunged through the liver lobe until the tip of the hemostat is seen penetrating through the opposite side of
the liver. The jaws of the hemostat are opened just wide enough to accept a piece of 2-0 or 3-0 Maxon or Biosyn suture. The suture is doubled on itself, the loop is passed into the jaws of the hemostats, and the loop pulled through the liver lobe. The exiting loop is cut leaving two strands of suture coursing through the liver lobe. Each strand is tied individually to “cut” through the liver. A V-shaped wedge is cut through the liver when both strands of suture have been tied. A number 15 BP scalpel blade is used to cut the V-shaped liver biopsy wedge from the sutures.

Pancreatic Biopsy*

Samples from the pancreas should be obtained in all suspected triaditis cases. The old wives’ tale stating “don’t touch the pancreas” needs to be put to rest in veterinary medicine. Gentle manipulation and biopsy of the pancreas is a predictably successful procedure with almost no incidence of postoperative pancreatitis. Biopsy of the pancreas is performed in a similar manner as biopsy of the liver. In patients that have diffuse pancreatic disease, a segment of the right or left limb of the pancreas is identified. An encircling ligature of 3-0 Biosyn is placed around the pedicle. As the ligature is tightened, it cuts through the pancreatic parenchyma, ligating vessels and pancreatic ducts. The distal pedicle of pancreas is carefully removed with a No. 15 BP scalpel blade or Metzenbaum scissors. Care is taken to avoid cutting the suture.

*A video demonstrating intestinal, liver, and pancreatic biopsy can be viewed at http://www.gloydgroup.com/proceedings/videos/2014-gi-symposium-surgical-abdominal-biopsy-feline. The video is provided courtesy of Dr. Howard B. Seim III.

TREATMENT OF IBD

It is important that the clinician formulate a treatment plan based on a correlation of clinical course, laboratory and gross findings, and histologic findings (considering both cellular infiltrate and morphology) rather than relying on histologic changes alone. Since food sensitivities can be a cause of IBD, dietary trials are an essential part of both the diagnostic and therapeutic strategy, utilizing hydrolyzed protein diets and novel protein diets and treating each patient as an individual (i.e., there can be variable responses to specific diets varying from patient to patient). Regarding pharmacotherapy, while corticosteroids have long been considered the cornerstone of treatment for idiopathic inflammatory bowel disorders (Table 2), antimicrobial agents may play a role as well. Bacteria have been implicated in the pathogenesis of IBD.

Pharmacotherapy

Guidelines for corticosteroids in cats with IBD are as follows. Mild to moderate cases of IBD often respond to prednisolone (preferred over prednisone in cats) at a starting dose of 1 to 2.2 mg/kg divided twice daily for 2 to 4 weeks followed by a gradual decline in 50% increments at 2-week intervals. Cats with inflammatory changes graded as mild usually respond quite well to the lower dose and alternate-day or every third day treatment can often be achieved by 2 to 3 months. Occasionally treatment can be discontinued altogether by 3 to 6 months.

If biopsies reveal disease that is moderate to severe a prednisolone dose of 2 to 4 mg/kg divided twice daily is used in cats for the first 2 to 8 weeks or until clinical signs resolve. This dose of corticosteroid is usually well tolerated in cats. In some cases a dose of 1 to 2 mg/kg per day may be necessary long term (months to years) to maintain clinical remission. Use of combination drug therapy may also be required at the outset to control clinical signs and prevent progression of the disease (e.g., metronidazole or tylosin plus prednisolone). Cats with hypoproteinemia and histologic changes graded as severe often respond quite well when an aggressive therapeutic course is undertaken.

Budesonide is a glucocorticoid that represents an alternative for management of IBD in dogs and cats, especially in severe cases that have proven to be refractory to prednisolone, metronidazole, azathioprine, chlorambucil, tylosin, and dietary management; or that are intolerant of the corticosteroids discussed above. Budesonide is one of a group of novel corticosteroids that have been in development for use in humans in an attempt to make available alternative preparations that will help limit toxicity associated with corticosteroid use.

Budesonide undergoes high first-pass metabolism in the liver and 90% is converted into metabolites with low corticosteroid activity. It has minimal systemic availability. The potential for typical corticosteroid side effects is significantly reduced as a result of decreased bioavailability and the resulting limited systemic exposure, which makes this a particularly attractive drug for use in humans and animals that are poorly tolerant of other corticosteroids. Budesonide also has a high receptor-binding affinity in the mucosa. It has been referred to as a “locally acting” corticosteroid. Therapeutic results with budesonide have been promising in humans with
Crohn’s disease, collagenous colitis, lymphocytic colitis, and ulcerative colitis, either when administered as a retention enema or in oral form, and primary biliary cirrhosis.

Budesonide has been used by some veterinary clinicians in recent years to treat IBD in dogs and cats. Dose recommendations vary. In humans, a range of 6 mg to 9 mg per day has been used during initial therapy. In general, budesonide is administered to cats at 1 mg administered once per day (this dose level is prepared at a compounding pharmacy).

Budesonide can be used in combination with other drugs. Since cats tolerate corticosteroids very well, there is little indication to use budesonide as initial therapy for IBD. However, this may be a very attractive option for use in diabetic cats that also have IBD, or in patients where conventional therapies have not been sufficiently effective.

Potential adverse effects include polyuria/polydipsia (PU/PD) when budesonide is used at the high end of the dose range and GI ulceration. These reactions have been observed in some human patients. These problems would be more likely to occur in dogs than in cats. It appears to be very safe when used at the levels listed above.

When combination therapy is indicated metronidazole is usually the first choice to be used in conjunction with prednisolone. Metronidazole’s mechanism of action includes an antiprotozoal effect, inhibition of cell-mediated immune responses, and anaerobic antibacterial activity. A dosage of 10 to 20 mg/kg two times daily is used for IBD. Ideally, at least several months of metronidazole therapy is given once it is started. In some cats with severe disease, long-term consecutive use or 1- to 2-month cycles of treatment may be required. Side effects of metronidazole at this low dose are uncommon in cats. Occasionally nausea or vomiting may be seen.

A See text for regimen details

Table 2. Pharmacotherapy of IBD in Cats

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dosage</th>
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| Prednisolone                | **Mild to moderate cases:** Starting dose of 1 to 2.2 mg/kg divided twice daily for 2 to 4 weeks followed by a gradual decline in 50% increments at 2-week intervals  
**Severe cases:** 2–4 mg/kg divided twice daily is used in cats for the first 2–8 weeks or until clinical signs resolve  
**Long-term (months to years) dose to maintain clinical remission:** 1–2 mg/kg per day |
| Budesonide                 | 1 mg administered once per day                                          |
| Metronidazole               | 10–20 mg/kg two times daily                                            |
| Methylprednisolone acetate | *Initially* 20 mg SC or IM; repeated at 2-week intervals for two to three doses.  
Then every 2–4 weeks or as needed for control |
| Chlorambucil               | *Initially* at 0.1–0.2 mg/kg/day in conjunction with prednisolone at 2.2 mg/kg/day  
Many clinicians use a starting dose of 1 mg per day or 2 mg every 48 hours |
| Cyclosporine               | 5 mg/kg once daily. Once sufficient response is achieved reduced to full dose every 48 hours and subsequently even further, on an individual patient basis |
| Cobalamin                  | 250 µg SC once a week for 6 weeks, then every 2 weeks for the next six doses; then dose monthly |

*a See text for regimen details*
If a client is unable to successfully administer oral medications, **methylprednisolone acetate** (Depo-Medrol) can be used as sole treatment for cats with mild to moderate IBD or as adjunctive therapy when oral prednisolone and/or metronidazole are used as the primary treatment and flare-ups of clinical signs occur. Consistent control of clinical signs in cats with moderate to severe IBD is more difficult to maintain when methylprednisolone acetate is used alone, however. It is recommended that sole use of methylprednisolone acetate be reserved for situations in which the owner is unable to consistently administer tablet or liquid prednisolone preparations. Initially 20 mg is given subcutaneously (SC) or intramuscularly (IM) and is repeated at 2-week intervals for two to three doses. Injections are then given every 2 to 4 weeks or as needed for control.

If remission cannot be maintained with use of corticosteroids and metronidazole then **chlorambucil** (Leukeran) should be used. Azathioprine was used more in the past but it has been largely supplanted now by chlorambucil. Chlorambucil is an alkylating agent. Alkylating agents alter DNA synthesis and inhibit rapidly proliferating cells. Chlorambucil is administered initially at 0.1 to 0.2 mg/kg/day in conjunction with prednisolone at 2.2 mg/kg/day. The small pill size of chlorambucil (2 mg) allows for easy dosing. Most cats receive half a tablet (1 mg) per day. Some clinicians initiate therapy at 2 mg every 48 hours. Toxicities are uncommon in cats but may include anorexia, vomiting, and diarrhea, but these problems generally resolve rapidly when chlorambucil is reduced from daily to every other day administration. Bone marrow suppression is possible but uncommon, and is mild and rapidly reversible when it does occur. Once the desired clinical response is achieved, chlorambucil is gradually tapered over several months while prednisolone is continued as the primary maintenance drug.

**Cyclosporine** is another immunosuppressive drug that can be used in management of IBD. Cyclosporine inactivates calcineurin phosphorylase in T cells, preventing transcription of interleukin-2 (IL-2) as well as other cytokines. Cyclosporine inhibits activation of T cells, natural killer cells, and Langerhans (i.e., antigen-presenting) cells. Suppression of the Th1 or Th2 response induces antigen tolerance. The dose is 5 mg/kg once daily. Once sufficient response is achieved the dosage interval can be reduced to administration of a full dose every 48 hours and subsequently reduced even further, on an individual patient basis.

**Cobalamin Therapy in Cats**

Significant tissue level cobalamin deficiency is present in some animals with GI disease. This is usually secondary to reduced cobalamin absorptive capacity. It is essential that all cats with any form of GI disease (including involvement of liver, stomach, pancreas, intestines) have a serum cobalamin level run to determine if the patient is hypocobalaminemic. Response to therapy will be limited if low cobalamin levels are not resolved. The reference range for cobalamin in cats is 290 to 1500 ng/L. Therapy is given if the value is less than 500 ng/L (i.e., in the low part of the reference interval; don’t wait until the level drops below the low end point of the reference range).

Therapy involves administering injectable cobalamin at the following schedule for cats: 250 µg SC once a week for 6 weeks, then every 2 weeks for the next six doses; then dose monthly. Most generic cobalamin preparations contain 1 mg/mL (1000 µg/mL). It is important to note that multi-vitamin and B-complex injectable formulations contain significantly lower concentrations of cobalamin and they also cause pain when injected. Therefore, it is recommended that these preparations not be used for cobalamin supplementation. Unless the intestinal disease is totally resolved, long-term and perhaps lifelong supplementation with cobalamin may be necessary. The frequency of injections on a long-term basis is determined by regular measurement of serum cobalamin concentration.

Because dietary allergens may play a role in the cause if IBD, specific dietary therapy may be beneficial. Often, moderate to severe degrees of IBD are either temporarily responsive or only minimally responsive to careful dietary manipulations. However, long-term control of IBD with as minimal a drug administration schedule as possible may be aided by specific dietary management. This should be started as soon as a diagnosis is made and continued as drug therapy is decreased later. Feed elimination (novel protein) or hydrolyzed protein diets. Chicken, duck, lamb, fish, or venison based diets are often tried initially. Elimination diets have been found to be very beneficial in cats.

Poor responses to treatment of cats with IBD usually result from:

1. Inadequate initial or long-term maintenance corticosteroid dosage in cats with more severe forms of IBD (moderate to severe disease)
2. Failure to use ancillary medications (metronidazole, chlorambucil, cyclosporine, tylosin) in cases where disease is moderate to severe
3. Failure to recognize and treat a concurrent condition (e.g., gastric hypomotility disorder that may either be secondary to IBD or idiopathic in nature, hyperthyroidism, parasitism [e.g., Giardia, Cryptosporidium], Clostridium perfringens enterotoxosis, cholangitis/cholangiohepatitis, chronic pancreatitis).

4. Treatment for only small intestinal inflammatory disease when colitis is present as well. Some cats with concurrent IBD and colitis may show minimal or no clinical signs of colitis.

5. Failure to recognize and treat low body cobalamin levels (measure serum cobalamin).

6. Failure to identify an effective diet.

7. Poor client compliance.

**What If Biopsies are Not Definitive for Either IBD or Small Cell Lymphoma?**

It can be difficult to definitively differentiate benign IBD from small cell intestinal lymphoma, even when full-thickness intestinal biopsies are obtained. If the biopsies were obtained via endoscopy, one option is to proceed to exploratory laparotomy to obtain full-thickness samples. However, this is not practical in some cases and involves a more invasive procedure and more expense. Further, there is no guarantee that the differentiation can be made even when full-thickness samples are obtained. Another option that is employed more commonly now is to perform special tests to help differentiate benign IBD from low-grade, small cell lymphocytic malignant lymphoma. Specific immunohistochemical techniques can be done to identify populations of malignant B and T lymphocytes (i.e., phenotyping) and molecular (PCR) testing is done for clonality. Clients should be given the option of ordering these additional tests if the pathologist indicates on the initial histopathology interpretation that the differentiation cannot be made definitively between IBD and lymphoma. If the client declines to have the additional tests performed, the clinician then needs to decide whether or not to just go ahead and treat for the disease that poses greater concern, i.e., lymphoma. Low grade small cell lymphoma is often treated with the combination of prednisolone and chlorambucil (see discussion on treatment details in the next section).

**TREATMENT OF INTESTINAL LYMPHOMA IN CATS**

Lymphoma is the most common feline neoplasm. It is also the most common form of gastrointestinal neoplasia in cats. Gastrointestinal lymphoma is often referred to as either well differentiated (low grade or lymphocytic), poorly differentiated (high grade, lymphoblastic, or immunoblastic), and intermediate (or mixed). Endoscopy has been shown to be a very useful modality for diagnosis of intestinal lymphoma in cats, especially when multiple biopsies are obtained using proper technique and instruments that can procure adequate size tissue samples. Immunohistochemical stains are beneficial for differentiating IBD from intestinal lymphoma in cases where it is difficult for the pathologist to distinguish between the two. Full-thickness intestinal biopsies may be required in a very limited number of cases in order to establish the correct diagnosis.

Many cats respond favorably to treatment for intestinal lymphoma, especially with the low grade or chronic lymphocytic type. Clinical signs can be very similar to cats with IBD. Therefore, it is strongly recommended that cats with chronic GI signs undergo a biopsy procedure as early as possible, so that the correct diagnosis can be established and the best course of therapy be made available for each individual cat. Biopsies should be obtained from both the upper and lower (ileum) small bowel.

Multi-agent chemotherapy is recommended for all cats with GI lymphoma. Surgery is done only if there is an isolated mass that is causing some degree of luminal obstruction. Survival times in excess of 12 to 18 months are not unusual. In some cats the response is somewhat shorter (3 to 6 months). The prognosis for longer survival time is much better if the diagnosis is made before clinical signs become chronic and debilitation results.

One study has reported excellent results in cats with chronic lymphocytic lymphoma using a protocol of prednisone (10 mg PO per cat per day) and chlorambucil (Leukeran) at a dosage of 15 mg/m² PO, once every day for 4 days, repeated every 3 weeks (Note: Prednisolone is used routinely at this time, rather than prednisone, in cats). Sixty-nine percent of the cats with lymphocytic lymphoma treated with this regimen achieved a complete remission. The median disease-free interval for cats that achieved complete remission was 20.5 months (range, 5.8–49 months). The median survival for all cats with lymphocytic lymphoma treated with chemotherapy was 17 months (range, 0.33–50 months). Cyclophosphamide (Cytoxan) was used for rescue in some of the cats that were entered in this protocol (225 mg/m², PO, every 3 weeks). For further reference on this protocol, see Richter K: Feline gastrointestinal lymphoma. ACVIM Proceedings, 2001, pp 547-549. Currently Dr. Tams uses a dose of 6 mg/m² for chlorambucil in
treatment of cats with small cell intestinal lymphoma, in conjunction with prednisolone.

The protocol that Dr. Tams has used most often for cats with the more aggressive lymphoblastic form of GI lymphoma was originally published by Cotter in 1983. Dosage levels have been modified slightly since that time. This protocol utilizes cyclophosphamide, vincristine (Oncovin), and prednisolone (COP). This protocol can be easily managed in any practice setting. Vincristine is administered intravenously at a dose of 0.5 to 0.75 mg/m² once weekly for 4 consecutive weeks and then once every 3 weeks. The initial doses are often decreased by approximately 25% for cats that are inappetent or debilitated. If well tolerated, the dose can then be gradually increased. Care is taken to ensure that none of the vincristine is given extravascularly. The average volume that is administered is quite low (0.1–0.15 mL for many cats, using a vincristine concentration of 1 mg/mL). Cyclophosphamide is given orally at a single dose of 225 mg/m² every 3 weeks (50 mg tablets are used with dosage adjusted to the nearest 25 mg on the low side of the calculated dose). Prednisolone is given orally at 10 mg per cat per day. Although cyclophosphamide and vincristine can be given on the same day, Dr. Tams often prefers to have the owner administer the cyclophosphamide 2 to 3 days after the oncovin. A CBC is done several times during the first month and then every 3 weeks to be sure that adequate granulocytes are present before treatment. At least 3,000 granulocytes/µL must be present before cyclophosphamide is given. If the granulocyte count drops to less than 1,000/µL 5 to 7 days after cyclophosphamide, the dose for subsequent treatments is reduced by 25%. The highest non-toxic dose is most likely to result in the greatest tumor cell kill.

The COP protocol is generally well tolerated, although side effects may occur and dosage or interval adjustments may be necessary. Side effects of COP in cats may include anorexia, vomiting, lethargy, and severe tissue irritation if any vincristine is given extravascularly. Also, the haircoat may become thinner, but complete hair loss does not occur. Cats do tend to lose whiskers. Cats should be carefully observed for sepsis especially during the induction phase. Prophylactic antibiotics are not indicated, but any infections that occur should be treated aggressively. Advantages of this protocol include hospital visits at only 3-week intervals after the first 4 weeks, lower cost to the owner, and a treatment interval that allows recovery of normal cells between treatments. Dr. Tams would like to emphasize that with careful monitoring and use of a dosage schedule that is tailored to each individual cat few problems are encountered. It is our general practice to encourage owners of most cats with GI lymphoma to pursue treatment that includes chemotherapy.

Nutritional and metabolic support is also important. If inappetence is a problem cyproheptadine can be administered as an appetite stimulant (1-2 mg orally every 12-24 hours) on an as needed basis (long-term if necessary). Mirtazapine is another appetite stimulant that can be used (one fourth of a 15-mg tablet every 3 days). Intermittent vomiting, nausea, and inappetence is managed with maropitant (Cerenia) administered at 4 mg orally for most cats once daily as long as it is needed. If there is concurrent renal disease with azotemia or if dehydration is a problem owners are taught how to administer subcutaneous fluids at home (e.g., lactated Ringer’s solution 100 to 150 mL every 24-48 hours, based on each individual cat’s needs). Special attention is given to ensuring that low cobalamin levels are addressed, if serum tests indicate that hypocobalaminemia is present. Occasionally chemotherapy can be discontinued after one year. This is done only if follow-up endoscopic intestinal biopsies indicate that there is no remaining lymphoma. Most cats remain on treatment for the remainder of their lives. If chemotherapy is poorly tolerated and reduced dosages and increased intervals between treatment times are unsuccessful in adequately decreasing side effects chemotherapy should be suspended. Prednisolone should be continued, however, because it may help maintain remission for a period of time. Doxorubicin (Adriamycin) can also be used in cats.

For clinicians inexperienced in administering chemotherapy, or who have not treated many cats with intestinal lymphoma, it is recommended that a veterinary oncologist or internist be consulted for guidance on protocol selection and ongoing management. Many cats with intestinal lymphoma can be managed successfully for some period of time!
REFERENCES


VIDEO – Intestinal Biopsy

VIDEO – Liver and Pancreatic Biopsy
Feline Intestinal Adenocarcinoma

David C. Twedt, DVM, DACVIM • David S. Biller, DVM, DACVR • Howard B. Seim III, DVM, DACVS

SUMMARY

Intestinal adenocarcinoma is the second most common gastrointestinal (GI) neoplasm occurring in the cat, representing approximately 25% to 30% of all GI tumors. The location is most often within the distal small intestine or proximal colon. Cats may have a long history of nonspecific GI disease that includes significant weight loss and vomiting. Imaging studies are key to the diagnosis and helpful tips for abdominal ultrasonography and contrast studies are provided in this article. Prognosis is guarded, but surgery (wide resection and anastomosis) can enhance quality of life and prolong lifespan.

CRITICAL KEYS

- Intestinal adenocarcinomas occur most frequently in cats in the distal small intestine or proximal colon.
- Weight loss is the hallmark of this condition because partial obstructions (or stagnant loop syndromes) often cause high levels of intestinal bacterial overgrowth that result in malabsorption.
- High serum folate and low serum cobalamin suggest small intestinal bacterial dysbiosis.
- Abdominal ultrasound is useful in identifying intramural intestinal lesions and upper GI contrast studies can show dilated loops of small intestine suggestive of ileus.
- Intestinal adenocarcinomas require wide resection and intestinal anastomosis. Even with clean regional lymph nodes and resection margins, however, metastasis is common and has often already occurred by the time of diagnosis.
- The overall prognosis for intestinal adenocarcinoma is guarded. A significant disease-free interval is possible, however, with some cats surviving for a year or longer, making surgery warranted.

The most common postoperative complication of small intestinal surgery is leakage. Leaks are associated either with breakdown of the anastomosis or improper surgical technique, and careful monitoring is critical.

The average age of cats with adenocarcinoma is greater than 10 years and there is a greater incidence in the Siamese breed. In retrospective studies some cats had a long history of nonspecific GI disease that included significant weight loss and vomiting. Frequently these tumors initially cause a distal partial small bowel obstruction resulting in a stagnant loop syndrome characterized by intestinal bacterial overgrowth proximal to the tumor. Weight loss is the hallmark of this condition with malabsorption.
due to the high intestinal bacterial numbers. Changes in intestinal mucosa, competition for nutrients, alterations in bile acids and pancreatic enzymes, and altered vitamin absorption (vitamin B12) from bacterial overgrowth result in significant weight loss often in spite of normal caloric intake. With a partial obstruction vomiting can also occur. In some cases the vomit may be quite fetid and in large volume from bacterial nutrient decomposition. Diarrhea may also occur in some cases.

On physical examination an abdominal mass associated with the intestine may be palpable; however, early in the disease a lesion may not be detected. Routine laboratory diagnostic testing is often unremarkable. Serum cobalamin (vitamin B12) is often low with an elevated folic acid. Serum trypsin-like immunoreactivity (TLI) concentrations are normal, ruling out pancreatic insufficiency. Imaging studies are the key to the diagnosis (see below).

**IMAGING STUDIES**

**Radiology**

Radiography is important for diagnosing intestinal diseases. During evaluation of the small bowel on survey radiographs, important factors that should be evaluated include:

- Location of small intestine (normally fills the abdomen where nothing else is present, not unusual to be mostly right-sided in cats)
- Appearance of bowel contents (gas, fluid, or mottled material),
- Contour of small bowel
- Diameter of the small intestine (the normal diameter in cats is up to 12 mm)

In normal animals, intestinal luminal contents should appear as a homogeneous fluid opacity. Disease of the small intestine may be missed on survey films unless there is a change in bowel opacity (mineralized mass or foreign material), luminal diameter (ileus or complete or partial mechanical obstruction), or changes in contour of the small bowel (linear foreign body).

Contrast studies (upper GI series) are often necessary to identify normal or abnormal shape, diameter, or continuity of small bowel. The transit time of barium varies greatly in cats. It usually travels from the stomach through to the ileum in about 60 minutes, although it can take as long as 4 hours.

The range of transit times for organic iodides through the small bowel is approximately 15 to 90 minutes. The organic iodide usually reaches the ileum and colon in less than 60 minutes.

Ileus is defined as an obstructive condition of the intestine that is either mechanical or functional. Mechanical ileus is also referred to as dynamic (or obstructive) ileus. It is usually simple and nonstrangulating. The radiographic signs may be influenced by the degree, location, and duration of the obstruction. Dilatation of small intestine secondary to mechanical obstruction results from swallowed air and saliva and accumulation of mucosal secretions in the digestive tract. Functional ileus, also referred to as “paralytic” (or adynamic) ileus, can be localized or generalized and may be secondary to mechanical ileus. The stages of development of functional ileus include muscle fatigue, allowing stretching of the intestine; muscle ischemia secondary to stretching; and muscle necrosis. Functional ileus has both extrinsic (which tend to be more generalized) and intrinsic (most often regional) causes. Extrinsic causes include spinal cord injury, reflex to pain, peritoneal trauma or irritation, or vascular compromise; intrinsic causes include edema, amyloidosis, and acute inflammation or enteritis.

**Ultrasoundography**

The small intestines can be seen throughout the abdomen, both end-on and longitudinally oriented. The duodenum has a slightly larger diameter than the rest of the small intestinal loops, and is the most lateral and ventral bowel loop in the right cranial abdomen. It usually can be located just ventral and lateral to the right kidney and followed cranially into the pylorus. The ileum has a distinct cross-sectional appearance (resembling spokes on a wheel) and can be visualized as it enters the colon, just medial to the right kidney. The colon typically is gas-filled, with poor visualization of the lumen.

The following five layers are present in the intestinal wall, from outside to inside:

- Serosa: Thin hyperechoic layer
- Muscularis: Thin hypoechoic layer
- Submucosa: Thin hyperechoic layer
- Mucosa: Prominent hypoechoic layer (typically the thickest layer)
- Mucosal surface–lumen interface: Hyperechoic layer in the center of the bowel
Normal wall thicknesses have been established in the cat for various segments of the GI tract:

- Duodenum: 2.0–2.4 mm (mean of 2.2 mm)
- Jejunum: 2.1–2.5 mm (mean of 2.3 mm)
- Ileum: 2.5–3.2 mm (mean of 2.8 mm)
- Colon: 1.4–1.7 mm (mean of 1.5 mm)

Ultrasonographic features of intestinal disease include bowel wall thickening, loss of wall layers, loss of motility, and regional lymph node involvement. One to three contractions per minute should be seen with normal small intestinal peristaltic activity.

**Adenocarcinoma**

Feline intestinal adenocarcinoma may have the following imaging findings. Radiographic findings may reveal a mid-abdominal mass associated with the GI tract and/or mesentery, localized or diffuse decrease, or loss of serosal detail suggestive of peritoneal effusion. If a mass is suspected radiographically or historically, or a mass has been palpated, then compression radiography may be helpful to isolate and visualize the mass. Obstruction, if present, often occurs gradually and resembles feces (mottled gas and a fluid material) is visualized in the small intestine. This can be mistakenly identified as colon. Contrast examinations (upper GI series) are helpful in identifying the mass or the obstruction. With contrast, the location and extent of the intestinal lesion may be more accurate than on survey images.

The most common ultrasonographic features of intestinal neoplasia are thickening of the bowel wall (>4 mm), loss of the normal layered appearance, reduced wall echogenicity, decreased localized motility, regional lymphadenopathy, and alterations in the contour of the mucosal and/or serosal surfaces. The most common ultrasonographic findings are solitary segmental concentric transmural thickening with a complete loss of layering that is often associated with lymphadenopathy. A thickened, asymmetric bowel wall, most commonly noted in the jejunum or ileum, may also be found. In many of these cases, there is evidence of fluid accumulation proximal to the intestinal thickening or mass associated with localized ileus. Localized abdominal effusion consistent with peritonitis or metastasis may be found. Metastasis may appear in regional lymph nodes as carcinomatosis (nodules) in the mesentery or omentum or in other abdominal organs, such as the spleen or liver.

**SURGERY***

Intestinal anastomosis is indicated for resection of a non-reducible intussusception, necrotic bowel wall secondary to complete intestinal obstruction, intestinal volvulus, stricture secondary to trauma, linear foreign body with multiple perforations, and intestinal neoplasia (e.g., leiomyoma, leiomyosarcoma, adenocarcinoma). After a complete abdominal exploration, the affected length of bowel is delivered from the peritoneal cavity and isolated with the use of moistened laparotomy pads and crib towels. If possible, the intestinal anastomosis should be performed on a water-resistant surface (e.g., plastic drape, crib towel) to prevent ‘strike-through’ contamination. Once the level of resection has been determined, the appropriate mesenteric vessels are identified and ligated, and the portion of intestine to be resected is isolated by clamping the bowel at a 60-degree angle away from the mesenteric border. This angle ensures adequate blood supply to the antimesenteric border.

When suturing an anastomosis, atraumatic handling of bowel wall and perfect anatomic apposition of incised margins is important. It is recommended to begin suturing at the mesenteric border as this allows adequate visualization of mesenteric vessels and helps prevent encircling these vessels when placing the first few sutures. Any of the appositional suture patterns previously described (i.e., simple continuous or interrupted) will result in a high success rate, both in the short term (i.e., leakage, breakdown) and long term (i.e., stricture, stenosis).

The following tips may prove helpful when performing an intestinal anastomosis:

1. First, place a stay suture to hold the mesenteric border of each segment of bowel in apposition. Tie this suture, leave the ends long, and place a hemostat on the suture end without the needle.
2. Place a second stay suture to hold the antimesenteric border of each segment of bowel and bring the ends of the intestinal segments into apposition. Place a hemostat on the ends of this suture.
3. Place gentle traction on the mesenteric and antimesenteric stay sutures to bring the two intestinal segments into apposition.
4. Using the needled segment of suture from the mesenteric stay suture, begin a simple continuous appositional anastomosis being careful to get a 3- to 4-mm bite in the submucosa and placing each suture no more than 2 mm apart (2 to 3 mm apart in dogs). When the
anastomosis is complete, tie the suture to the mesenteric stay suture.

5. If a simple interrupted apposing suture pattern is used, be careful to get a 3-mm bite in the submucosa and place each suture no more than 2 to 3 mm apart.

The author’s (H.S.) preference for evaluating the integrity of anastomotic closure is to visually examine each suture to be certain that suture placement is no more than 2 to 3 mm apart and that each suture has a 3-mm bite in the submucosa.

Occasionally when the segment of intestine to be removed is amputated mucosa ‘everts’ from the cut edge of the intestinal wall making it difficult to visualize the cut edge of the serosa. If this occurs it is ‘highly’ recommended to excise the everted mucosa to enable the surgeon to easily visualize the cut edge of the intestinal serosa. It is vital that the surgeon engage at least 3 to 4 mm of intestinal wall with each suture to guarantee adequate bites in the collagen laden submucosa.

In cases where the oral end of the bowel is dilated and the aboral end is normal size, several options exist to create intestinal lumens of equal diameter:

1. Increase the angle of resection on the smaller diameter segment of bowel (i.e., aboral segment). This will increase the orifice size by 5 to 10 mm depending upon bowel diameter (e.g., dog vs. cat).

2. In larger lumen size discrepancies the antimesenteric border of the smaller diameter stoma can be incised longitudinally to enlarge the lumen diameter.

3. An endto-side anastomosis can be performed by closing the larger diameter stoma of the intestinal resection with a single layer continuous apposing suture pattern, then anastomosing the smaller diameter segment of bowel to an appropriate size enterotomy made in the antimesenteric border of the larger diameter segment of bowel.

4. The larger diameter segment of bowel can be made smaller in diameter by suturing its cut edge until its lumen is equal in size to the smaller diameter intestine (this technique is often used for subtotal colectomy in cats).

*A video demonstrating intestinal anastomosis in a cat with intestinal adenocarcinoma can be viewed at http://www.gloydgroup.com/proceedings/videos/2014-gi-symposium-intestinal-anastomosis-feline. The video is provided courtesy of Dr. Howard B. Seim III.*

**POSTOPERATIVE CARE**

Intravenous fluids are given to maintain hydration, ensure renal function, and to correct electrolyte changes. Systemic antibiotics are continued postoperatively for 5 to 7 days or 10 to 14 days in cases with peritonitis and/or sepsis. Early return to enteral feeding is best for the overall health of the intestine. In some cases enteral feeding tubes may be required. Vitamin B12 is supplemented based on serum concentrations (or empirically at 250 µg subcutaneously, SC, weekly). The sooner patients can be returned to oral alimentation the better.

The most common postoperative complication of small intestinal surgery is leakage. Leaks are either associated with breakdown of the anastomosis or improper surgical technique (i.e., improper suture placement, inappropriate suture material, knot failure, sutures to far apart, inappropriate bite in the collagen laden submucosal layer, suturing nonviable bowel). Careful monitoring is critical to detect leakage.

**PROGNOSIS**

The overall prognosis for intestinal adenocarcinoma is guarded. Even with clean regional lymph nodes and resection margins metastasis is common. With wide intestinal resection and anastomosis, median survival time was reported to be 2.5 months (range 0–24 months) in one study. Tubular adenocarcinomas may have a better prognosis than other histologic types, especially if metastasis is not present at the time of surgery. A significant disease-free interval is possible, however, with some cats surviving for a year or longer, making surgery warranted.
SUGGESTED READING


VIDEO – Intestinal Anastomosis in Cat with Intestinal Adenocarcinoma