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CHAPTER 31

Diseases of the Stomach

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CONGENITAL/DEVELOPMENTAL DISORDERS

Pyloric Stenosis

Definition

I. It is a congenital disease of boxers and Boston terriers.
II. Stenosis of the pyloric canal occurs from hypertrophy of the pyloric circular muscle.

Causes

I. The cause is unknown, but an oversecretion of gastrin has been postulated.
II. A functional abnormality has also been proposed, as some animals with pyloric outflow obstruction do not have muscular thickening.

Pathophysiology

I. Stenosis of the pyloric canal causes gastric outflow obstruction.
II. Elevated levels of gastrin can lead to thickening of the pyloric smooth muscle from its trophic effects.
III. Gastrin can also lead to hypertrophy of the mucosa, which can worsen the outflow obstruction.

Clinical Signs

I. Clinical signs are related to delayed gastric emptying.
II. Generally, animals vomit food >12 hours after eating.
    A. The vomiting may be explosive.
    B. The food in the vomitus is usually digested, but may appear undigested.
III. Abdominal distention may be noted.
IV. Weight loss may occur from inability to retain food.
V. Anorexia is uncommon.

Diagnosis

I. Pyloric outflow obstruction is suspected based on the pattern of vomiting.
II. Laboratory findings may show hypokalemia, hypochloremia, and metabolic alkalosis.
III. Plain radiography often shows a stomach distended with gas and fluid.
IV. Contrast radiography may show delayed gastric emptying and a narrowing of the pyloric canal that is referred to as the *beak sign*.
V. Nuclear scintigraphy may also be used to identify delayed gastric emptying.
VI. Abdominal ultrasonography can be used to detect thickening of the pylorus.
VII. Endoscopy may be normal if the mucosa is not thickened.
VIII. At surgery, the pylorus is palpably thickened, and gastrotomy reveals a thickening of the muscular layer.

Differential Diagnosis

I. Other causes of mechanical pyloric outflow obstruction: mucosal hypertrophy, intraluminal masses (e.g., polyps, neoplasia, foreign bodies)
II. Causes of a functional delay in emptying: electrolyte disorders, pain, peritonitis, acute pancreatitis, dysautonomia, gastric ulceration

Treatment and Monitoring

I. After the animal has been stabilized with appropriate fluid and electrolyte therapy, the definitive treatment is surgery.
II. Surgical procedures include pyloromyotomy or various pyloroplasty techniques.
III. Prognosis following adequate surgical correction is good.

INFECTIOUS DISORDERS

Parasitic Gastritis

Definition and Causes

I. Parasites affecting the stomach of cats include *Ollulanus tricuspis* and *Physaloptera* spp. (less common).
II. Gastric parasites of dogs include *Physaloptera* spp.
III. Parasites attach to the gastric mucosa, causing inflammation and gastritis.

Clinical Signs

I. Infections in both dogs and cats may be inapparent.
II. Intermittent vomiting is the most common clinical sign.
III. Variable anorexia may also occur.

Diagnosis

I. *Ollulanus* spp. can be difficult to diagnose.
    A. The parasites and eggs are usually missed on routine parasitologic (usually not passed in the feces) and endoscopic (very small in size) examinations.

B. The best method of diagnosis is microscopic examination of the vomitus.
C. Organisms may also be seen on histopathology.
D. Chronic hypertrophic fibrosing gastritis may also be seen on histopathology.

II. Diagnosis of *Physaloptera* spp. is a little easier.
A. Eggs may be found on fecal flotation, but may not be routinely isolated.
B. The parasites can be seen with endoscopy and appear as 1- to 4-cm–long, white worms attached to the stomach.

Differential Diagnosis

I. Other causes of acute gastritis (see below)
II. Causes of chronic and secondary gastritis

Treatment and Monitoring

I. *Ollulanus* spp.: fenbendazole 10 mg/kg PO SID for 3 days
II. *Physaloptera* spp.: pyrantel pamoate 5 mg/kg PO for two doses, 3 weeks apart
III. Prognosis is excellent for recovery after treatment.

Helicobacter Gastritis

Definition

I. *Helicobacter* gastritis is inflammation of the stomach caused by various species of the genus *Helicobacter*.
II. Many cats and dogs that have colonization of the stomach with these spiral organisms do not have concurrent inflammation.

Causes

I. The organisms are spiral-shaped, gram-negative, motile bacteria.
II. Various species have been isolated from the stomachs of cats, dogs, ferrets, cheetahs, and others.
III. *Helicobacter heilmannii* and *Helicobacter felis* are the most common species that naturally occur in dogs and cats.
IV. *Helicobacter pylori* has been found in laboratory cats, but not in dogs.
V. These bacteria may be normal inhabitants of the stomach.

Pathophysiology

I. The pathophysiology of this disease is unclear.
II. The organisms produce urease (urea $\rightarrow NH_3 + HCO_3$) that buffers acid and allows colonization of the superficial mucus and gastric glands of the stomach.
III. The bacteria have been observed intracellularly.
IV. In infected animals, degeneration of gastric glands, as well as vacuolation and necrosis of parietal cells, has been seen.
A. The inflammation is generally mononuclear (lymphocytes, plasma cells) and can vary in the degree of severity.
B. Lymphoid follicle hyperplasia can be seen.
V. Infected animals may have up-regulation of various cytokines.

Clinical Signs

I. Chronic vomiting is the most common sign.
II. Diarrhea, anorexia, pica, and polyphagia have also been reported.
III. Uncommonly, fever and/or bloody diarrhea may be seen.

Diagnosis

I. Laboratory tests are usually normal, but may show nonspecific changes (e.g., stress leukogram).
II. Abdominal radiography and ultrasonography are usually normal.
III. The best method of diagnosis currently is endoscopic biopsy.
A. Organisms can be visualized on the epithelium or in the mucus layer.
B. Warthin-Starry silver stains enhance visualization of the organisms.
C. Multiple biopsy samples are taken as the colonization can be patchy.
D. Endoscopy also allows evaluation of the mucosa for the type and severity of inflammation and for other changes.
IV. The rapid urease test may be performed and is based on the production of urease by almost all *Helicobacter* spp.
A. The test is also known as the *Campylobacter*-like organism (CLO) test.
B. Gastric tissue is incubated in broth with a pH indicator (phenol red) so that a color change indicates production of ammonia via urease.
C. The degree of color change is proportional to the density of organisms.
D. Results are available in 1 to 24 hours.
E. This test has a sensitivity of 70% to 90% (Happonen et al., 1996).
V. Brush cytology of gastric mucus is a relatively sensitive method of detecting organisms but does not allow determination of whether inflammation is present.
VI. Culture is not usually performed because the organisms are hard to grow in the laboratory.
VII. The C13 urea breath test also has been used in dogs and cats; however, it is not widely available.
VIII. Polymerase chain reaction (PCR) assays of gastric tissue allows diagnosis as well as identification of the species present; however, it is not widely available.
IX. Serological testing is widely used as a screening test in humans, but tests designed for humans should not be used in dogs and cats because the primary organism affecting humans (*H. pylori*) is not generally found in dogs or cats.
X. Serological testing is also difficult in animals because multiple species of *Helicobacter* may occur.

Differential Diagnosis

I. Other causes of chronic vomiting: chronic gastritis, pyloric outflow obstruction
II. Vomiting caused by nongastrointestinal diseases

Treatment

I. The best therapy has yet to be identified in the dog and cat.
II. "Triple therapy" is recommended in symptomatic humans and involves administration of an acid-inhibiting drug, bismuth compounds, and an antibiotic (e.g., clarithromycin, amoxicillin).
III. Most of the therapies tried in dogs and cats have not been 100% successful; many animals, although initially cleared of the organism, become reinfected.
IV. Combinations that have been tried include the following:
  A. Metronidazole, amoxicillin, and famotidine
  B. Azithromycin, tinidazole, bismuth, and ranitidine
  C. Clarithromycin, metronidazole, bismuth, and ranitidine
  D. Amoxicillin, metronidazole, and omeprazole
  E. Amoxicillin, metronidazole, and clarithromycin
V. Therapy is usually given for 2 to 3 weeks.

Monitoring of Animal

I. Monitoring is generally via physical examination and clinical signs.
II. To definitively determine if the organisms have been cleared, invasive testing may need to be repeated, but this is usually not done if the clinical signs resolve.

Gastric Pythiosis

Definition and Cause

I. Gastric pythiosis in dogs is caused by the aquatic oomycete *Pythium insidiosum*.
II. It has been documented only as a cutaneous and subcutaneous infection in cats; however, unpublished information indicates it can occur rarely as a gastrointestinal (GI) infection in cats.
III. The organism may dwell in water or soil.
IV. In the United States, the infection occurs most commonly in the Gulf Coast states, but it has been recognized as far north as New Jersey and Illinois, and as far west as Oklahoma, Missouri, and Kansas.

Pathophysiology

I. Pythiosis occurs most commonly in young, male, large-breed dogs—especially outdoor, working dogs.
  A. Affected dogs are usually exposed to areas of warm, fresh water.
  B. The animals are usually immunocompetent.
II. The infective form is likely the zoospore and may cause infection by encysting in damaged GI mucosa.
III. Infection is characterized by severe transmural thickening of the stomach, and the gastric outflow tract is one of the most common sites of infection.
IV. Inflammation is usually in the submucosa, with variable mucosal ulceration.
V. The disease may extend through to the serosal surface, and associated lymph nodes may be enlarged.

Clinical Signs

I. Severe weight loss and vomiting are common.
II. Lethargy is not usually seen unless obstruction has occurred.
III. Diarrhea and hematochezia may be seen when other parts of the GI tract are involved.

Diagnosis

I. Laboratory abnormalities may include eosinophilia, anemia, hyperglobulinemia, and hypoalbuminemia.
II. Abdominal radiography and ultrasonography reveal thickening of the gastric (usually pyloric) wall.
III. Associated lymphadenopathy also is usually seen on ultrasonography.
IV. Definitive diagnosis requires identification of the organism.
V. Cytology of aspirates of enlarged lymph nodes or thickened stomach wall shows pyogranulomatous, suppurative, or eosinophilic inflammation, but the organism is seen only occasionally.
VI. A presumptive diagnosis can be made based on histopathology.
  A. Findings include granulomatous, eosinophilic to pyogranulomatous inflammation with fibrosis.
  B. Organisms are usually found in the center of granulomas or in areas of necrosis.
  C. The organisms are easier to visualize with Gomori's methamine silver (GMS) staining.
  D. Immunohistochemistry techniques may also be used.
VII. Culture of the organism is difficult unless special sample handling and culture techniques are used.
VIII. A serological enzyme-linked immunosorbent assay (ELISA) has been developed for the detection of antibodies and is highly sensitive and specific in dogs and cats.
IX. Western immunoblot analysis also can be used.

Differential Diagnosis

I. Gastric neoplasia
II. Hypertrophic gastritis
III. Other systemic fungal infections.

Treatment

I. Aggressive surgical resection (3- to 4-cm margins) of the infected area is the treatment of choice.
  A. The organisms may not be present in enlarged lymph nodes, so they are not routinely resected.
  B. Medical therapy for 2 to 3 months (as follows) is recommended following resection because of the possibility of recurrence.
II. If resection is not possible, antifungal therapy can be tried.
  A. Response is often poor, but up to 15% of dogs may respond (Grooters, 2003).
  B. Itraconazole 10 mg/kg PO SID and terbinafine 5 to 10 mg/kg PO are recommended for 6 to 9 months.
  C. Alternatively, amphotericin B lipid complex is given at 2 to 3 mg/kg IV QOD up to a cumulative dose of 24 to 27 mg/kg.

INFLAMMATORY DISORDERS

Acute Gastritis

Definition

I. It is inflammation of the stomach that has an acute onset.
II. It implies that the cause and the inflammation can be eliminated, so that the stomach returns to normal health, with no residual inflammation or fibrosis.

Causes and Pathophysiology

I. Ingestion of pathogenic bacteria rarely cause gastritis because they are usually unable to colonize the stomach.
II. The main exception is the spiral bacteria (*Helicobacter* spp., see *Helicobacter* gastritis).
III. Viruses (e.g., parvovirus, distemper virus, coronavirus) may cause gastritis as part of a more widespread condition.
IV. Bacterial toxins produced by *Clostridium* spp., *Escherichia coli*, and *Klebsiella* spp. have been suggested.
V. Physical damage from foreign bodies and thermal injury can result in gastritis.
VI. Chemicals, such as cleaning agents, floor finishes, and various plant toxins, are also potential causes.
VII. Certain drugs (aspirin) may be directly cytotoxic to the stomach, whereas others exert their toxic effects by indirect mechanisms.
VIII. Garbage ingestion is a common cause of acute gastritis in dogs.
IX. Many metabolic diseases cause gastritis (e.g., renal and hepatic failure, hypoadrenocorticism).

Clinical Signs

I. Acute vomiting is the most common clinical sign, especially after eating or drinking.
II. Blood is occasionally present in vomitus.
III. Varying degrees of anorexia, depression, and abdominal pain may also be noted.

Diagnosis

I. Tentative diagnosis is based on the history and clinical signs.
II. Definitive diagnosis is not commonly made, because animals usually recover rapidly.
III. Laboratory tests are usually unremarkable.
IV. Abdominal radiographs are usually unremarkable, unless an opaque foreign body is present.

Differential Diagnosis

I. Causes of chronic or secondary gastritis: gastrinoma, mast cell tumor, renal failure, hepatic failure
II. Nongastric diseases that cause vomiting: pancreatitis, hypoadrenocorticism

Treatment

I. Administer fluid therapy if needed.
  A. A balanced electrolyte solution is adequate.
  B. Give fluids SC or IV, depending on the severity of the clinical signs.
II. Stop all oral intake (nothing by mouth [NPO]) for 12 to 36 hours.
III. Reintroduce bland food as small, frequent meals.
  A. The food should contain an easily and highly digestible starch, be low in protein, and contain moderate to low fat.
  B. Several commercial canine and feline formulations are on the market and most are labeled as "intestinal formulas."
  C. Homemade diets for dogs include boiled hamburger and rice, low-fat cottage cheese and rice, chicken and rice, cooked egg whites and rice, or tofu and rice, all in ratios of 1:2 or 1:4.
  D. Homemade diets for cats include chicken or turkey, possibly combined with baby rice cereal in a 1:1 ratio.
IV. Antiemetics are used when necessary; they are only symptomatic therapies, so are not to be used in place of adequate diagnostic and specific therapies.
  A. They are reserved for intractable vomiting when the cause of the vomiting is known.
  B. They vary in their site(s) of action.
    1. Alpha$_2$-adrenergic antagonists (phenothiazines)
      a. The sites of action are the chemoreceptor trigger zone (CRTZ) and the vomiting center.
      b. These are potent antiemetics and are effective for most causes of vomiting.
      c. Side effects include sedation and hypotension.
      d. Examples for both dogs and cats include prochlorperazine *(Compazine)* 0.5 mg/kg IM, SC TID and chlorpromazine *(Thorazine)* 0.2 to 0.4 mg/kg SC TID.
    2. Histaminergic (H_1) antagonists (antihistamines)
      a. Sites of action are the CRTZ and the vestibular apparatus.
      b. Side effects include sedation.
      c. These are usually given only for motion sickness.
      d. Examples in the dog include diphenhydramine *(Benadryl)* 2 to 4 mg/kg PO, IM TID and dimenhydrinate *(Dramamine)* 4 to 8 g/kg PO TID.
    3. Dopaminergic ($D_{2)}$ antagonists
      a. Sites of action include the CRTZ and GI smooth muscle.
      b. They are used for vomiting secondary to uremia, delayed gastric emptying, etc.
      c. Side effects include extrapyramidal signs (from metoclopramide) and sedation (from haloperidol).
      d. Examples include metoclopramide *(Reglan)* 0.2 to 0.4 mg/kg PO, SC, IM TID to QID, or 1 to 2 mg/kg/day IV as a constant rate infusion (dog, cat) and haloperidol *(Haldol)* 0.02 mg/kg PO BID (dog).
    4. Serotonergic 5HT$_3$ antagonists
      a. Sites of action are the CRTZ and vagal afferent neurons.
      b. They are used primarily during chemotherapy.
      c. Side effects include sedation.
      d. An example in the dog is ondansetron *(Zofran)* 0.5 to 1.0 mg/kg PO SID to BID or 30 minutes before chemotherapy.

5. Serotonergic $5HT_4$ agonists
   a. Sites of action are the myenteric neurons.
   b. They are used primarily for delayed gastric emptying, rather than as an antiemetic.
   c. An example is cisapride *(Propulsid)* 0.1 to 0.5 mg/kg PO BID to TID (dog, cat).
6. Motilin agonists
   a. Site of action is the GI smooth muscle (motilin receptors).
   b. They are used for delayed gastric emptying, rather than as an antiemetic.
   c. Side effects include vomiting.
   d. An example in the dog is erythromycin 0.5 to 1 mg/kg PO, IV TID.

V. Inhibition of gastric acid secretion is usually not needed; however, H_2 blockers may be used if gastric bleeding is noted (see next section).
VI. Antibiotics are not indicated unless a specific bacterial pathogen is suspected or documented, or disruption of the gastric mucosal barrier is significant.
VII. Locally acting protectants (sucralfate, bismuth subsalicylate) are usually not needed, but may be used safely if desired (see next section).

Gastric Ulceration and Erosion

Definition and Causes

I. Ulceration may be acute and caused by chemicals, gastric dilatation-volvulus, drugs, disseminated intravascular coagulation (DIC), shock, or foreign bodies.
II. Ulcers may arise with chronic disorders, such as inflammatory bowel disease (IBD), neoplasia, renal failure (acute or chronic), and hepatic failure.
III. The most common causes include nonsteroidal anti-inflammatory drugs (NSAIDs), neoplasia, shock, renal and hepatic failure, and hypoadrenocorticism.

Pathophysiology

I. NSAIDs inhibit prostaglandin production, which results in loss of an important part of the gastric mucosal barrier.
   A. Certain drugs (aspirin) can be directly cytotoxic to the epithelial cells.
   B. Risk factors for ulcer formation include higher doses, long-term administration, and concurrent administration with another NSAID or a corticosteroid.
II. Certain neoplasms commonly cause gastric ulceration.
   A. Mast cell tumors release histamine, which causes an increase in H^+ production.
   B. Gastrinomas release gastrin, which also results in increased H^+ production.
III. Shock results in disruption of blood flow to the stomach, leading to ischemia and ulceration.
IV. Renal and hepatic failure causes abnormal metabolism of gastrin (among other substances), with increased production of H^+.
V. In the preceeding situations, reparative mechanisms of the mucosa are overwhelmed and superficial damage (erosions) occurs, or severe lesions that penetrate to the muscularis or deeper (ulcers) develop.

Clinical Signs

I. Clinical signs are variable, with vomiting being the most common.
II. The vomitus may contain fresh or digested blood ("coffee grounds").
III. Anorexia may be noted.
IV. Abdominal pain can occur that may be ameliorated by food (from buffering action of food).
V. Animals may develop acute abdominal pain from gastric perforation and peritonitis, with few or no prior clinical signs.

Diagnosis

I. Laboratory findings may be normal.
II. The cause of ulceration may be detected by laboratory findings (e.g., azotemia, hepatic failure).
III. Acute or chronic microcytic, hypochromic anemia may be seen.
IV. Abdominal ultrasonography reveals changes in the gastric wall consistent with ulceration and focal or diffuse accumulation of peritoneal fluid if a perforation is present.
V. Any free abdominal fluid may be sampled with ultrasound guidance.
VI. Definitive diagnosis requires visualization of the ulceration/erosion via endoscopy.

Differential Diagnosis

I. Other causes of vomiting
II. Other causes of GI bleeding and melena, including thrombocytopenia and other clotting disorders

Treatment

I. Treat any underlying conditions and stabilize the animal with appropriate fluid therapy, electrolyte replacement, and transfusion therapy as necessary.
II. If the animal is receiving a drug that may cause an ulceration, discontinue the drug.
III. Institute specific treatment with antacids.
   A. Acid neutralizers
      1. These agents neutralize acid that has already been produced by the stomach.
      2. They can be very effective, but must be given at least six times per day.
      3. Examples include magnesium hydroxide *(Milk of Magnesia),* aluminum hydroxide *(Amphojel),* and calcium carbonate *(Tums).*
      4. Aluminum-containing antacids decrease absorption of phosphorus and may stimulate mucosal defense mechanisms.
      5. Calcium-containing antacids may cause constipation.
   B. H_2-receptor antagonists
      1. These agents selectively and reversibly bind to H_2 receptors on the oxyntic cell, thus inhibiting the acid secretagogue effect of histamine.

2. Although these drugs only partially inhibit acid secretion, they often allow healing to occur.
3. Cimetidine (5 to 10 mg/kg PO, SC TID to QID in dogs and cats) reversibly inhibits the hepatic microenzyme system (cytochrome P-450), and can interfere with clearance of drugs metabolized by this route.
4. Ranitidine may be six to ten times more potent than cimetidine.
   a. Ranitidine inhibits microsomal enzymes less than cimetidine.
   b. Ranitidine may have gastric prokinetic properties.
   c. Dose is 1 to 4 mg/kg PO, SC, IV BID to TID (dogs and cats).
5. Famotidine has potency similar to ranitidine in dogs, with a longer elimination half-life.
   a. Dose in dogs is 0.5 mg/kg PO, SC, IV SID to BID
   b. Dose is not established for cats.

C. Proton pump antagonist
1. These drugs irreversibly inhibit the hydrogen-potassium-ATPase pump at the apical border of the oxyntic cells.
2. A single daily dose results in virtual antacidity.
3. They also inhibit hepatic microsomal enzymes similar to cimetidine.
4. They are superior to H_2 blockers for treatment of severe reflux esophagitis and indolent gastroduodenal ulceration in dogs.
5. Dose of omeprazole is 0.7 to 2 mg/kg PO SID in dogs; very little experience exists in cats.

IV. Gastric protectants are also useful.
A. Misoprostol is a synthetic prostaglandin E1 analog that inhibits gastric acid secretion and stimulates gastric mucosal defense mechanisms in dogs.
1. Its primary therapeutic use is prophylaxis against gastric mucosal injury caused by NSAIDS.
2. Its main adverse effect is diarrhea.
3. Do *not* use it in pregnant animals, and do *not* allow pregnant women to handle it (can cause abortion).
4. Dose in dogs is 1 to 5 μg/kg PO BID to TID.

B. Sucralfate is a complex salt of sucrose sulfate and aluminum hydroxide.
1. In an acidic environment, sucralfate binds to exposed submucosa and polymerizes.
2. Its primary action is to stimulate mucosal defense and reparative mechanisms, as well as inhibit pepsin activity.
3. Sucralfate stimulates bicarbonate and mucus secretion, increases the viscosity of gastric mucus, and stimulates the release of prostaglandins (facilitates mucosal blood flow and repair).
4. It is not absorbed from the GI tract, but may inhibit absorption of other drugs.
5. It may cause constipation.
6. Dose is 0.25 to 1 g PO BID to QID in dogs and 0.125 to 0.25 g PO BID to TID in cats.

C. Bismuth subsalicylate *(Pepto-Bismol)* has cytoprotective properties by complexing with glycoproteins to retard hydrogen ion diffusion through the mucosa and by decreasing pepsin output.
1. It also has antibacterial activity.
2. Dose in dogs is 0.5 to 1 mL/kg PO every 4 to 8 hours.
3. It must be used with caution in cats because of the salicylate component (0.25 mL/kg PO BID).

V. Antiemetics are used as needed (see Acute Gastritis).
VI. Prokinetic agents enhance GI motility, specifically gastric emptying.
A. Metoclopramide has antiemetic and prokinetic properties.
1. It increases gastroesophageal sphincter (GES) pressure and hastens gastric emptying.
2. Dose in both dogs and cats is 0.2 to 0.4 mg/kg PO, SC, IM TID to QID or 2 mg/kg/day IV as a constant rate infusion.

B. Cisapride has prokinetic and antiemetic effects, but is used primarily as a prokinetic agent.
1. It increases GES pressure, accelerates gastric emptying, enhances colonic propulsive motility, and probably enhances motility of the small intestine.
2. Dose in both dogs and cats is 0.1 to 0.5 mg/kg PO BID to TID.
3. Cisapride is available through a few compounding pharmacies.

C. Erythromycin has an action similar to endogenous motilin, as well as an antiemetic action.
1. It accelerates gastric emptying and has intestinal promotility affects.
2. Dose is 0.5 to 1 mg/kg PO, IV TID in dogs.

D. Ranitidine and nizatidine may stimulate gastric, intestinal, and colonic motility, but their clinical efficacy is unknown.

VII. Uncommonly, gastric ulcers may bleed profusely or may be so deep that perforation is imminent, making partial gastrectomy and resection of the affected area necessary.

Monitoring of Animal

I. Laboratory tests are repeated to ensure that electrolytes have normalized and anemia (if present) is stable or improving.
II. Endoscopy can be repeated to determine definitively if the ulceration has healed, but is not usually done unless clinical signs persist.

Chronic Idiopathic Gastritis

Definition and Causes

I. It is defined as chronic inflammation of the stomach where no cause can be found.
II. The condition may be immune-mediated, and is considered a form of IBD that is localized to the stomach.
III. Postulated causes include food allergy, loss of tolerance to bacterial (normal flora) antigens, genetic susceptibility, or an abnormal immune response (host hypersensitivity).

Pathophysiology

I. The pathophysiology of IBD is complex and poorly understood.
II. Chronic gastritis is a diagnosis of exclusion.
III. Histopathologic findings include occasional microerosions of the epithelium, infiltration of the interstitium with lymphocytes, plasma cells or eosinophils.
  A. Fibrosis of gastric tissue is seen after prolonged, untreated inflammation.
  B. Lesions can be patchy or diffuse.
IV. In cats, eosinophilic gastritis may be seen as part of a more generalized disease (hypereosinophilic syndrome).

Clinical Signs

I. Vomiting is the most common clinical sign.
II. Mild weight loss is seen less commonly.
III. Variable anorexia and depression may occur.

Diagnosis

I. Laboratory tests are usually normal or show nonspecific changes (e.g., stress leukogram, eosinophilia).
II. Abdominal radiographs are usually normal.
III. Abdominal ultrasonography may show a thickened stomach wall and/or enlargement of gastric lymph nodes.
IV. Known causes of gastritis must be excluded.
  A. Negative fecal examination and no response to deworming
  B. No history of NSAID use
V. Gastric biopsy is necessary for definitive diagnosis.
  A. Endoscopic biopsies are often adequate.
  B. Biopsies also may be obtained via exploratory laparotomy.
VI. At endoscopy, the mucosa may appear grossly normal, granular, friable, hyperemic, edematous, or eroded.

Differential Diagnosis

I. Other causes of primary gastritis: food intolerance, parasites, foreign body
II. Other causes of secondary gastritis: renal or hepatic failure, hypoadrenocorticism

Treatment

I. Treatment often involves a combination of dietary changes and/or drug therapy.
II. Feed small, frequent meals of a low-fiber, low-to-moderate–fat diet to hasten gastric emptying.
III. Diets can be of three types.
  A. An easily and highly digestible commercial diet or a home-cooked diet can be prepared.
  B. A novel protein diet can be tried.
  C. A hypoallergenic diet consisting of hydrolyzed protein sources can be tried.
  D. The dietary change may be effective alone, or may be used in combination with drug therapy.
IV. Antiinflammatory or immunosuppressive drugs are indicated if nutritional management alone does not control the clinical signs.
  A. Give prednisone at 2 mg/kg PO SID for dogs and 2 to 4 mg/kg PO SID for cats for 2 to 4 weeks, then gradually tapered over 3 to 6 months.
  B. Some animals may be weaned completely off corticosteroids, whereas others must remain on chronic low doses (usually 0.5 to 1 mg/kg PO QOD).
V. If the animal is refractory to steroid therapy, relapses, or has unacceptable side effects, alternative drugs may be needed.
  A. Metronidazole may be helpful.
  B. Other options include azathioprine or cyclosporine in dogs and chlorambucil or cyclosporine in cats.
VI. Mucosal protectants are used as needed if erosions are present.
VII. Inhibition of gastric acid secretion with H_2 blockers or a proton pump inhibitor may be beneficial.
VIII. Prokinetic agents may be of value if delayed gastric emptying is a concurrent problem.

Monitoring of Animal

I. Repeat laboratory tests to ensure that the animal is stable.
  A. Some animals with severe idiopathic gastritis may have gastric bleeding, so the monitor the packed cell volume (PCV).
  B. Some animals may develop side effects (e.g., diabetes mellitus) secondary to corticosteroid use, so monitor blood glucose as well.
II. Repeat endoscopy and biopsy to definitively determine if the inflammation is under control; however, they are not usually done unless clinical signs persist.

Atrophic Gastritis

Definition and Causes

I. It is a very uncommon condition.
II. The etiology is unknown, but it may be immune-mediated or the terminal stage of idiopathic gastritis.

Pathophysiology

I. The pathophysiology is unknown.
II. Histopathology shows a reduced gastric mucosal parenchyma (loss of glands and cells), some inflammatory cells (lymphocytes and plasma cells), flattened epithelium, shortened gastric pits, metaplastic cells, and fibrosis.
III. Achlorhydria (loss of ability to produce H^+) often results and leads to small intestinal bacterial overgrowth, which in turn may lead to malabsorption.

Clinical Signs

I. Intermittent vomiting is the most common clinical sign.
II. Mild weight loss may be seen, and anorexia and depression may occur.

Diagnosis

I. Laboratory tests are usually normal, but may show nonspecific changes (e.g., stress leukogram).
II. Abdominal radiographs and ultrasonography are usually normal.

III. Endoscopy with biopsy is the method of choice for diagnosis.
IV. The mucosa may appear grossly normal or discolored and thin (more common), with submucosal blood vessels visible under the mucosa.

Differential Diagnosis

I. Other GI causes of vomiting: food intolerance, parasites, foreign body
II. Non-GI causes of vomiting: renal or hepatic failure, hypoadrenocorticism

Treatment

I. Change the diet to small, frequent meals of an easily digested diet, or try a novel protein diet.
II. Use mucosal protectants if erosions are present.
III. Inhibition of gastric acid secretion may make clinical signs worse if achlorhydria is present.
IV. Prokinetic agents are of value if delayed gastric emptying is a problem.
V. Antiinflammatory drugs are indicated if nutritional management alone does not control the clinical signs.
   A. Prednisone is given at 1 to 2 mg/kg PO SID for 2 to 3 weeks then gradually tapered over 3 to 6 months.
   B. Additional antiinflammatory agents are uncommonly needed.

Monitoring of Animal

I. Laboratory monitoring is done for side effects secondary to corticosteroid use (e.g., blood glucose, alkaline phosphatase).
II. Endoscopy and biopsy can be repeated to determine definitively if the inflammation is under control, but are not usually done unless clinical signs persist.

Hypertrophic Gastritis

Definition

I. Hypertrophic gastritis is characterized by focal or diffuse mucosal proliferation along with inflammation.
II. In the focal form, polypoid lesions may occur.
III. Widespread mucosal thickening is less common.

Causes

I. The etiology of this uncommon condition is unknown.
II. Genetics may play a role because it is more common in small-breed dogs (e.g., Lhasa apso, shih tzu, Maltese, basenji).
III. An immune-mediated cause has also been postulated.
IV. Male dogs are predisposed.

Pathophysiology

I. The pathophysiology is unknown, but hypergastrinemia may be involved, as gastrin is trophic to the gastric mucosa.
II. Histopathology shows hypertrophy and hyperplasia of the mucosa, metaplasia of glandular epithelium, and variable amounts of fibrous tissue and inflammatory cells (lymphocytes, plasma cells).

Clinical Signs

I. Intermittent vomiting is the most common clinical sign.
II. Mild weight loss may be seen with variable anorexia and depression.

Diagnosis

I. Laboratory tests and abdominal radiographs are usually normal or show nonspecific changes (e.g., stress leukogram).
II. On endoscopy the mucosa is diffusely or focally thickened, usually in the area of the antrum, and biopsy confirms the diagnosis.
III. Measurement of serum gastrin concentration is done to rule out a gastrin-secreting tumor.

Differential Diagnosis

I. Other GI causes of vomiting: food intolerance, parasites, foreign body.
II. Non-GI causes of vomiting: renal or hepatic failure, hypoadrenocorticism.

Treatment

I. Change the diet to small, frequent meals of an easily digested diet, or try a novel protein diet.
II. Mucosal protectants may be beneficial if erosions are present.
III. Inhibition of gastric acid secretion with H_2 blockers or a proton pump inhibitor may be helpful.
IV. Prokinetic agents are of value if delayed gastric emptying from a motility problem is present, but they are indicated if hypertrophied mucosa causes a physical obstruction.
V. Prednisone may be tried (1 to 2 mg/kg PO SID for 2 to 3 weeks, then gradually tapered over 3 to 6 months) if inflammation is present and nutritional management does not control the signs.
VI. Surgical resection of focal areas of hypertrophy is performed, especially when bleeding polypoid lesions or gastric outflow obstruction are present.

Monitoring of Animal

I. Laboratory monitoring is done for side effects secondary to corticosteroid use (e.g., blood glucose, alkaline phosphatase).
II. Endoscopy and biopsy can be repeated to determine definitively if the inflammation is under control, but they are not usually done unless clinical signs persist.

MISCELLANEOUS DISORDERS

Delayed Gastric Emptying

Definition

I. Food is retained in the stomach for an abnormally long time.
II. The stomach is usually completely empty within 10 to 12 hours after a normal meal.

Causes

I. Various primary intestinal diseases can result in delayed gastric emptying.
   A. Mechanical obstruction from gastric mucosal hypertrophy, pyloric muscular stenosis, foreign bodies, polyps, neoplasia, pythiosis
   B. Functional obstruction or motility disorders from acute or chronic gastritis, acute pancreatitis, gastric ulceration, gastric neoplasia
II. Delayed gastric emptying may also be secondary to non-GI disease.
   A. Metabolic acidosis, electrolyte disorders (hyper- or hypocalcemia, hypokalemia)
   B. Diabetes mellitus, pain, peritonitis, trauma, abdominal surgery
   C. Drugs (narcotics), dysautonomia, hypoadrenocorticism, hepatic failure, uremia

Pathophysiology

I. Pathophysiology depends on the underlying cause.
II. Mechanical obstruction is a physical impedance to outflow of contents from the stomach.
III. Functional obstruction arises from an alteration in normal gastric motility causing defective gastric propulsion, and may be related to an abnormality of neuronal or smooth muscle function or coordination.

Clinical Signs

I. Acute or chronic vomiting is the most common clinical sign.
   A. Whether the vomiting is acute or chronic depends on the underlying condition.
   B. The vomiting often occurs long after ingestion of the meal at a time when the stomach would normally be empty (>10 to 12 hours after eating).
   C. The vomiting may be explosive or projectile.
II. Abdominal discomfort is sometimes noted.
III. Anorexia and depression are uncommon.
IV. Weight loss may be seen with chronic disease.
V. Various other clinical signs are seen depending on the underlying cause, such as polyuria and polydipsia with diabetes mellitus and renal failure.

Diagnosis

I. Regardless of the cause, laboratory tests often shows hypochloremic metabolic alkalosis secondary to loss or pooling of HCl in the stomach.
II. Other laboratory changes depend on the underlying cause (e.g., hyperglycemia with diabetes mellitus, azotemia with renal failure).
III. Contrast radiography is one of the best methods to identify a mechanical outflow obstruction.
IV. Fluoroscopy is also helpful when a functional disorder is suspected.
V. Endoscopy can be used to help determine the cause of a mechanical obstruction, but it is not very helpful if a functional problem is present.

Differential Diagnosis

I. Other causes of acute and chronic vomiting
II. Motility problems of the intestines

Treatment and Monitoring

I. Certain foreign bodies can be removed via endoscopy, while others require surgical removal.
II. Surgery is the treatment of choice for antral pyloric muscular hypertrophy, certain cases of mucosal hypertrophy, pythiosis, and other causes of mechanical obstruction.
III. The underlying cause must be treated in cases of functional obstruction.
   A. Insulin therapy and electrolyte replacement for diabetes mellitus
   B. Antiinflammatory medications for chronic gastritis
IV. Prokinetic agents often are very helpful for functional disorders (see Treatment under Gastric Ulceration and Erosion).
V. Endoscopy and biopsy, ultrasound, or fluoroscopy with contrast media can be repeated to definitively determine if the disease is under control, but these tests are not usually performed unless clinical signs persist.

Bilious Vomiting Syndrome

Definition and Causes

I. Bilious vomiting syndrome is an idiopathic disorder associated with duodenogastric reflux of bile.
II. Duodenogastric reflux stimulates the vomiting reflex.

Clinical Signs

I. Dogs tend to vomit small amounts of bile first thing in the morning on an empty stomach.
II. The physical examination is usually normal.

Diagnosis and Differential Diagnosis

I. Diagnosis is suggested based on the pattern of vomiting and lack of other clinical signs.
II. Laboratory tests are normal.
III. Definitive diagnosis is by exclusion of other causes of chronic vomiting.

Treatment and Monitoring

I. A small meal given just before bed often helps.
II. A prokinetic drug at bedtime may be added if needed.
III. Prognosis is good to excellent for control of the condition.

NEOPLASIA

Definition and Causes

I. Adenocarcinoma is the most common gastric neoplasm of the dog, and lymphoma is the most common tumor of the cat.
II. Other tumors affecting the stomach include the fibrosarcoma, leiomyoma, leiomyosarcoma, and plasmacytoma.

Pathophysiology

I. Neoplastic cells infiltrate the stomach in either a focal (adenocarcinoma) or diffuse (lymphoma) pattern, and may be mucosal or transmural.
II. Mucosal ulceration is common.
III. Lymphoma in cats has been described as large or small cell in type.

Clinical Signs

I. Chronic vomiting, weight loss, and inappetence are the most common clinical signs.
II. Vomitus may include old blood (coffee grounds appearance), and melena may be seen in some cases.

Diagnosis

I. Laboratory tests may be normal or show nonspecific changes (e.g., stress leukogram).
   A. Anemia may be seen with bleeding tumors.
   B. The anemia may be acute and nonregenerative, or chronic (microcytic, hypochromic).
II. Survey abdominal radiographs may reveal a mass effect or gastric wall thickening.
   A. Positive contrast techniques or pneumogastrography may be helpful (see Chapter 4).
   B. Both techniques are performed *after* abdominal ultrasonography, because they interfere with visualization of the stomach and other organs.
III. Abdominal ultrasonography may reveal a gastric mass, enlarged lymph nodes, or evidence of metastasis (liver).
IV. Definitive diagnosis requires biopsy and histopathology.
V. Endoscopy may be performed to obtain a biopsy diagnosis, but does not determine whether metastasis is present.
VI. Surgery can be performed for diagnostic purposes, as well as for therapy.

Differential Diagnosis

I. Other causes of chronic vomiting: GI and non-GI in origin
II. Other neoplasia of the GI track and abdomen

Treatment and Monitoring

I. Treatment depends on the tumor type.
II. The recommended therapy for lymphoma is multi-drug chemotherapy (see Chapter 69).
   A. Remission times are much shorter for dogs than cats.
   B. Small-cell lymphoma in cats is usually treated with prednisone (or prednisolone) and chlorambucil.
      1. Prednisone is started at 5 mg PO BID and chlorambucil at 15 mg/m^2 PO SID for 4 days and repeated every 3 weeks.
      2. An alternative regimen is to give chlorambucil at 6 mg/m^2 PO QOD.
      3. Long-term remissions may be achieved in these cats.
III. Recommended therapy for other types of gastric neoplasia is surgical resection.
   A. The primary mass is resected and >2- to 3-cm margins are included if possible.
   B. With adenocarcinomas, large margins are difficult and metastases are often present at the time of diagnosis.
   C. Prognosis may be better for leiomyosarcoma if the mass is resectable.
   D. Adjunctive chemotherapy has not been shown be beneficial for most gastric tumors, although carboplatin and doxorubicin may be alternated every 3 weeks for three treatments each.
   E. Median survival times are often short (approximately 4 months).

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