

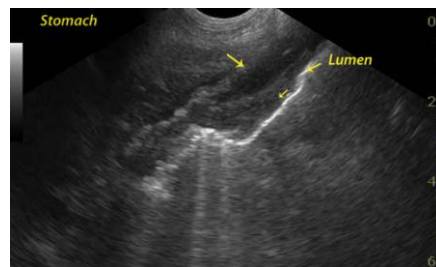
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Animal Sounds

MARCH 2012
CANINE EROSIVE GASTRITIS AND DUODENITIS:
HEMATEMESIS AND MELENA – 4 PAGES



Dr. Lauren Costa & I bring you the latest on a disease process that we, at NJ Mobile and SonoPath.com are currently performing a study on regarding the sonographic aspects or erosive Gi disease. The simple anorexic +/- vomiting patient may have something like this brewing that needs special attention and treatment that may be surgical before peritonitis develops. The sonogram is the best way to assess mural GI disease in small animals.

Erosive gastritis patients often do not vomit, but are simply **anorexic** and **depressed**, have a **decreased appetite**, are **middle aged to older**, have a **low normal or anemic HCT**, have a history of **weight loss**, unexplained **BUN elevation** due to GI blood loss, and often a history of NSAID or high dose steroid administration. A worse case scenario includes a pneumoabdomen due to ulcer perforation and peritonitis. Patients may display **hematemesis** (“coffee ground” or large amounts of frank blood in vomitus), and/or **melena** (pitch black tarry stool that when placed on white paper, leaves a bloody residue). Melena, however, is only seen if there is an acute loss of a lot of blood into the upper gastrointestinal tract. Common causes of chronic, unresolving gastrointestinal ulceration/erosion are: 1) mast cell tumor (can cause gastric acid hypersecretion), 2) drug administration, 3) stress

(a substantial decrease in GI perfusion such as hypovolemic shock, systemic inflammatory response syndrome, neurogenic shock), 4) hepatic failure, 5) gastric tumors (leiomyoma, leiomyosarcoma), 6) hypoadrenocorticism, and

7) gastrinomas (small pancreatic tumors which produce large amounts of gastrin which in turn causes gastric acid secretion). Ultrasound examination is typically recommended as a first choice diagnostic, and aids in determining whether guided biopsy, endoscopy or surgical evaluation need be pursued to attain a definitive diagnosis. **Research has demonstrated repeatedly that NSAIDs cause ulceration in virtually all dogs, even the newer “safer” choices.** Not all patients, however, show clinical signs of the condition.

The clinical work up for GI ulcerations/erosions should include a platelet count and coagulation profile (to rule out a coagulopathy), abdominal ultrasonography to rule out the presence of a gastrointestinal mass (also helps to detect mural abnormalities), gastroduodenoscopy, and surgery (if a ulceration/erosion is managed appropriately without significant improvement or is bleeding so terribly that it can wait for medical management). Endoscopy can be extremely diagnostic and help in detecting gastric erosions, ulcerations, hemorrhages, lymphoid follicle hyperplasia (mucosal pock marks), and increased or decreased mucosal friability. For examples, large amount of bile-stained fluid in the stomach could indicate duodenogastric reflux-associated gastritis. When biopsies are taken via endoscopy, at least three biopsy samples should be obtained from each region of the stomach (pylorus, fundus, and cardia).

Gastric and duodenal thickening can be due to acute or chronic inflammatory origin, parasitic in origin or due to infiltrative neoplasia. Neoplastic G.I. presentations typically display diminished peristalsis in the area of question, loss of mural detail (ill-defined muosa, submucosa, muscularis, serosa), and have pronounced hypertrophy (>0.8-1.4 cm serosa to mucosal thickness) on ultrasound examination. These criteria do not *define* neoplasia but merely *suggest* it. Acute inflammatory presentations will usually have less dramatic changes in thickness, edematous but defined mural layers (unless ulcers are present) and hyperperistalsis (unless aperistalsis is present due to systemic disease). Chronic inflammatory states, such as that of chronic food intolerance/IBD, can present a mixed bag of these cited features. Ulcerative and polypoid changes are typical with mural fibrosis and secondary pyloric stenosis. Wall thicknesses are variable and can exceed 2 cm and often satisfy malignant criteria. For this reason tissue biopsy is essential for rapid diagnosis, and can be obtained endoscopically. All of the above pathologies often present stasis of intraluminal fluid displaying variable echogenicity due to excessive acid secretion, outflow obstruction and delayed gastric emptying.

Signs of **gastrointestinal neoplasia** often present rapidly and carry a poor prognosis (4-6 mos. for lymphoma, adenocarcinoma, with chemo +/- sx, better for leiomyosarcoma), as the disease progresses and infiltrates the pyloric outflow tract. Interestingly, although leiomyoma/sarcoma carry better prognoses, these masses often ulcerate easily and early, and cause significant hematemesis. Full thickness biopsies are best for thorough diagnosis given that endoscopic sampling is mucosal/submucosal. Inflammation, hyperemia, or normal mucosa can be seen in even in cases of intramural neoplasia. However, endoscopy is often diagnostic if the pathology penetrates superficially as well as helpful in finding/removing unseen foreign bodies and evaluating ulcerative changes. Ultrasound guided biopsies are often attainable; fasting and sedation may allow for better evaluation and availability of a sonographic window. Gastrinomas of the alimentary tract are also of a concern, causing *Zollinger-Ellinson Syndrome*; ulceration due to excessive gastrin production from a tumor of the pancreas or other tissue. Typically multiple duodenal erosions or small ulcers are noted, or there is a large ulcer located just distal to the pylorus. Mast cell disease often manifests itself with gastrointestinal inflammation or infiltration, with ulcer distribution often similar to that seen in gastrinoma cases. Screening for MCT externally on PE and internally by ultrasound is essential. Ultrasound guided biopsy is often possible if an adequate acoustic window and adequate wall thickness is available. Endoscopy will allow for biopsies in cases with mucosal/submucosal defects and allow for evaluation of overall pathology. Laparotomy or laparoscopy with full thickness biopsies of the GI and adjacent organs (liver, lymph node) frequently ends up being necessary in cases of neoplasia where the lesion is not reachable via ultrasound or endoscopy.

Other less common causes of gastroesophageal ulcerative disease include **hepatic failure** and **hypoperfusion**-related “stress” to the gastric wall. Foreign bodies themselves do not cause ulcers, however a patient with a foreign body and concurrent ulcer is at risk of perforation. Hematemesis is also seen with cases of **hypoadrenocorticism** and **heavy metal toxicity**. It is important to remember that hematemesis as a clinical sign, does not always denote gastric bleeding, but can be caused by swallowed blood. Failure to detect erosions or ulcerations on endoscopy in such cases requires thorough evaluation of the nasal passages, upper airways and thorax to evaluate for presence of lesions, which may be causing hemorrhage. In addition, it is prudent to obtain a **coagulation profile** on patients displaying signs of gi blood loss to avoid missing a coagulopathy and as a prerequisite for biopsy.

Treatment

Treatment is aimed at treating the underlying metabolic disorders and removal of drugs, toxins, foreign bodies, parasites, and fungal infections.

- 1) A highly digestible and low residue (low in insoluble fiber) **therapeutic diet** such as Royal Canin HE, Hill's Science Diet i/d, or Purina EN is recommended. Feeding method is important and multiple, small meals are recommended (> 4 feedings/day) in order to prevent excessive retrograde chyme mixing and prevent moderate to excessive gastric fill. If a chronic history is present consider a hypoallergenic food trial to treat potential underlying food intolerance.
- 2) **Sucralfate** (protects areas where ulceration and erosion are already present) at 1 gram/30kg PO BID-TID; Loading dose of 1-2 g PO q2-4 hours if actively bleeding and then decrease to 1-2 PO TID (dose size dependant) in moderate/severe cases. Give 1-3 hrs after other medications due to absorptive interference (Fluoroquinolones, tetracycline, amino/theophylline, digoxin..) **Sucralfate should be dissolved in a small amount of water and given as a solution. Side effects—constipation.**
- 3) **H₂ Receptor antagonists** (Cimetidine, Ranitidine, Famotidine)—decrease the gastric hydrogen ion concentrations; used to treat existing ulcerations/erosions. Do not prevent them.
 - Famotidine (Pepcid) 0.5 mg/kg PO, IV, IM, SID/BID (H₂ Blocker)
 - Ranitidine (Zantac) 2 mg/kg PO, IV, IM, SC, BID
 - Cimetidine (Tagamet) 5-10 mg/kg PO, IV, SC, TID/QID (Watch hepatic drug interference!)
- 4) **Proton Pump Inhibitors** (Omeprazole, Pantoprazole, Lansoprazole)—most effective at inhibiting gastric acid secretion; generally take 2 to 5 days to reach maximum efficacy.
 - Omeprazole (Prilosec) 0.7-1.5 mg/kg PO SID (typical dose 0.7 mg/kg PO SID)
 - Lansoprazole (Prevacid) 1 mg/kg
 - Pantoprazole—not approved for usage in dogs.
- 7) **Misoprostol** (Cytotec) 2-5 u/kg PO TID (Prostaglandin analog great for NSAID related ulcers or as concurrent therapy with chronic NSAIDs)
- 8) **Antacids** up to 6x daily (Magnesium hydroxide)
- 9) **Antibiotic therapy** for Helicobacter-Associated Gastritis
 - Combination of metronidazole, amoxicillin, and Famotidine in dogs for approximately 14 days. A recent study showed that longer treatment (21 days) lead to better improvement in the eradication of Helicobacter.
- 10) **Parasiticides**—if the gastritis is secondary to parasites (Ollulanus, Physaloptera)
 - Fenbendazole administered for 5 consecutive days.
- 11) I/D, Low Residue or **hypoallergenic food trial**, in a slurry if pyloric outflow obstruction/hypertrophy is significant.
- 11) **Pain management:** no NSAIDS
- 12) Options for **antiemetics** include Cerenia (maropitant) at 1 mg/kg SC SID or 2 mg/kg PO SID for up to 5 days, Zofran (ondansetron) 0.11-0.22 mg/kg IV/IM/SC or Anzemet (dolasetron) at 0.2-0.6 mg/kg IV SID or metoclopramide at 0.2-0.4 mg/kg PO or SC TID.

A combination approach including an H₂ receptor blocker, antacid, antiemetic, and dietary adjustment is a good first line treatment plan. Omeprazole is used for biopsy confirmed refractive cases or severe esophagitis, and misoprostol is the first line choice when treating gastritis secondary to NSAIDs.

If a 3-week trial therapy post ultrasound exam proves unfruitful, or if the patient clinically declines despite therapy, tissue sampling is essential to determine the underlying pathology.

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