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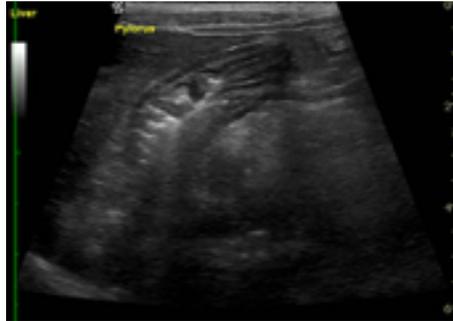


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**FEBRUARY 2014**

**CANINE EROSIVE GASTRITIS AND DUODENITIS: HEMATEMESIS AND MELENA**



**Description:** Erosive gastritis can have multiple causes, and patients often have a **history of NSAID or high dose steroid administration**. Research has demonstrated repeatedly that NSAIDs cause **ulceration** in virtually all dogs, including the newer, “safer” choices. Not all patients, however, show clinical signs of the condition. Other causes include **inflammatory conditions** such as inflammatory bowel disease, infiltrative or primary neoplasia. **Gastric neoplasia** can include lymphoma, adenocarcinoma, gastrinoma, and sarcoma, and carries a poor prognosis of 4-6 months. **Mast cell disease** can also cause gastric ulceration, manifesting with GI inflammation or infiltration. **Helicobacter gastritis** is a much-debated topic in veterinary medicine, but the organisms are frequently located within gastric ulcerations and are related to ulcerative disease in humans. Other, less common causes of gastrointestinal ulcerative disease include **hepatic failure** and stress to the gastric wall secondary to **hypoperfusion**. **Heavy metal toxicity** and **hypoadrenocorticism** can also cause gastric ulceration. Finally, **coagulopathy** and **upper airway or thoracic disease** can cause hematemesis but not gastric ulceration, and should be pursued if a primary cause for GI bleeding is not apparent.

**Clinical Signs:** Erosive gastritis patients **often do not vomit**, but are simply **anorexic** and **depressed**. These patients tend to be **middle aged to older**. Patients may display **hematemesis** (either “coffee grounds” or large amounts of frank blood in vomitus), and/or **melena** (black, tarry stool that, when placed on white paper, leaves a bloody residue). **Pneumoabdomen** can occur in severe cases secondary to ulcer perforation and peritonitis. Signs of **gastrointestinal neoplasia** often present rapidly, and interestingly, the neoplasias which carry a slightly better prognosis (leiomyoma/sarcoma) often ulcerates easily and early, causing significant hematemesis. 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.

**Diagnostics:** A minimum database including complete blood count, biochemical profile, and urinalysis may demonstrate **low normal or anemic hematocrit**, and unexplained **BUN elevation** due to GI blood loss. **Ultrasonographic examination** is typically recommended as a first choice imaging diagnostic, and aids in determining whether ultrasound-guided biopsy, endoscopy or surgical evaluation need be pursued to attain a definitive diagnosis. **Tissue biopsy** is essential for rapid diagnosis, and can be obtained endoscopically. However, since endoscopic biopsies include only mucosa and submucosa, full thickness biopsies are preferred for definitive diagnosis. Renal failure and uremic gastritis can also be an underlying cause of GI irritation. Hence, if clinical parameters consistent with renal failure are present then uremic gastritis should be at the forefront of the diagnostic differential.

**Treatment:** **Diet** should include a highly digestible and low residue (low in insoluble fiber) therapeutic diet. Feeding method is important and **multiple, small meals** are recommended. Four or more feedings per day is adequate to prevent excessive retrograde chyme mixing and prevent moderate to excessive gastric filling. **If pyloric outflow obstruction** is suspected or confirmed, food may need to be fed in **slurry** form. **Buprenorphine** (0.005 - 0.02 mg/kg IM, IV or SC q6-12H) can provide adequate control of mild to moderate pain. **Anti-emetics** may include maropitant (1 mg/kg SC q24H or 2 mg/kg PO q24H for up to five days), ondansetron (0.11 - 0.22 mg/kg IV, IM or SC q24H), dolasetron (0.2 - 0.6 mg/kg IV q24H) or metoclopramide (0.2 - 0.4 mg/kg PO or SC TID).

**Sucralfate** (1 gram/30 kg) PO BID-TID can be dissolved into a small amount of water and administered as a slurry. In the face of active bleeding, a loading dose of 1-2 grams given orally every 2-4 hours can be used, then decreased to 1-2 grams orally TID depending on the size of the dog. Sucralfate should be given 1-3 hours after other medications due to absorptive interference with other drugs. **H2 receptor antagonists** including famotidine (0.5 mg/kg PO, IV, IM q24H-BID), ranitidine (2 mg/kg PO, IV, IM, SC BID) or cimetidine (5-10 mg/kg PO, IV, SC TID/QID) should be given. **Alternatively, a proton pump inhibitor** can be used (omeprazole 0.7 - 1.5 mg/kg PO q24HR). The prostaglandin E1 analog **misoprostol** (2-5 mcg/kg PO TID) is especially helpful in treating and preventing gastric ulceration secondary to NSAID use. **Antacids such as magnesium hydroxide** can be used up to q4 hours.

If **Helicobacter spp.** is suspected, **erythromycin** (10-15 mg/kg TID) or **doxycycline** (10 mg/kg BID) can be considered and should be administered for 2 weeks. **Metronidazole** should be added along with an anti-secretory agent (H2 antagonists or PPIs, at doses listed above) to either antibiotic for immune palliation, triple *Helicobacter* therapy, and to target anaerobic bacteria.

**Conclusion:** A combination approach including an H<sub>2</sub> receptor blocker, antacid, antiemetic, and dietary adjustment is a good first line treatment plan. Omeprazole is used for histopathologically confirmed refractive cases, and misoprostol is the first line choice when treating gastritis secondary to NSAIDs.

**References:**

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