GI TRACT ULCERATION/EROSION AND GI BLEEDING

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Abstract
GI bleeding is not always obvious. Ulcers are one cause, but many dogs with ulcers do not bleed. NSAIDs are still an important cause.

Introduction
Most of the time, hematemesis is denoted by a “coffee-grounds”-like material that most clients (and some veterinarians) do not recognize as blood. A common mistake is being concerned over “dark stools”. Noting that a patient has dark stools is generally useless. Lots of dogs have dark stools and no problems or GI blood loss at all. The color of the stool is not an issue until the stool is pitch-tar-coal-asphalt black. Then it may be melena (if it is not due to Bismuth). If in doubt, just place some fresh feces on absorbent white paper and see if a reddish color diffuses out from the feces, confirming that there is blood present. Melena is only seen if there is acute loss of a lot of blood into the upper GI tract. Most dogs losing blood in the upper GI tract do not have any important changes in the color of the feces. Fecal occult blood tests are seldom that helpful or necessary, but can occasionally be informative in confusing cases. There tend to be 3 major reasons: coagulopathy, swallowing blood from elsewhere and gastrointestinal ulceration/erosion (GUE).

Coagulopathies
Most coagulopathies cause concomitant bleeding from the nose or accumulation of blood in body cavities or petechia. However, there are many cases in which the only sign of a systemic coagulopathies is GI bleeding.

Ulcers and erosions
The most common causes of chronic, unresolved GUE that are also the easiest to check for are mast cell tumor, drug administration and “stress”.

Drugs: These are still a very important cause of GUE in the dog. High doses of dexamethasone also have
substantial potential for significant GUE. Combining steroids and non-steroidal drugs can be devastating, but you can use ultra-low dose aspirin (0.5 mg/kg once daily) when treating IMHA dogs with steroids.

All NSAIDs have the potential to cause devastating GUE. While the newer Cox-2 NSAIDs (e.g., carprofen, etogesic, deracoxib, meloxicam, etc) have much less potential for causing GUE than the older NSAIDs, you can still see GUE (and even perforation) due to these drugs.

**Stress:** When mentioned as a cause of ulcers, stress specifically refers to substantial decrease in visceral perfusion (e.g., hypovolemic shock, neurogenic shock, Systemic Inflammatory Response Syndrome).

**Mast cell tumors:** These tumors may look like any skin lesion. When these tumors degranulate, they release histamine which if of sufficient magnitude can cause gastric acid hypersecretion. This can result in severe ulceration, especially just inside or just beyond the pylorus. **Hepatic failure** seems to be another important cause of GUE in the dog. Anytime a dog with hepatic disease suddenly becomes clinically worse (especially if it becomes obviously encephalopathic), you should consider the possibility of GUE. **Gastric tumors** may cause bleeding. The leiomyoma and leiomyosarcoma in particular may cause especially dramatic bleeding. **Surgery** can be responsible for GI bleeding. If the closure is done improperly and the mucosa does not cover the defect, then bleeding can easily result.

**Hypoadrenocorticism** may be responsible for severe hematemesis that can produce life-threatening shock. Such severe hematemesis appears to be a rare complication of hypoadrenocorticism. **Gastrinomas** are typically small pancreatic tumors which produce large quantities of gastrin, a hormone which causes gastric acid secretion. Multifocal duodenal ulceration/erosion is very suggestive of this tumor, as is a large ulcer just past the pylorus (as for mast cell tumors). **Foreign objects** get a lot of press as causes of GUE, but in fact they are relatively uncommon causes. However, they are particularly important in patients that have GUE because even the most innocuous of GI foreign objects (e.g., paper, small piece of soft cloth) can sometimes prevent a pre-existing ulcer from healing. They typically need to be removed in patients with GUE.

**Ingesting blood:** It is surprisingly easy to have bleeding pulmonary lesions in which the blood is coughed up, swallowed, and later vomited.

**Clinical approach to patients with hematemesis or GI bleeding** First eliminate coagulopathy with a platelet count and some measure of clotting factor adequacy. A mucosal bleeding time is a very useful screening test in these patients. After coagulopathy has been eliminated, then imaging should be done if it has not already occurred, and ultrasound is especially important as it may reveal masses that can be aspirated percutaneously, thus avoiding the need for endoscopy/surgery. If these tests have not revealed the diagnosis, then gastroduodenoscopy is generally performed next. The specific reasons to do gastroduodenoscopy in a patient with GI bleeding are to:

a) determine if this is a case in which surgery can remove a defined number of ulcers (this is for cases that are bleeding and have not responded to medical management or cases that are bleeding so badly that one cannot wait on medical management). In these cases, it is important to be sure that bleeding is not due to widespread erosions that cannot be cured surgically. There is no relationship between the
size of the mucosal defect and the amount of bleeding; patients with lots of small erosions often bleed as bad or worse than patients with ulcers. It is also important to determine the number and location of such ulcers as they may be hard to find during a gastrotomy.
b) determine if there is a gastric tumor or some other infiltrate in a patient with GUE that is non-responsive to appropriate therapy.
c) determine the cause in a patient with GUE and no apparent cause on the history, physical examination, or routine blood work.
d) look for a cause of bleeding in a patient with GI blood loss of unknown cause.

Only by treating and observing the patient will you know if an ulcer will or will not respond to medical management. If there is substantial upper GI blood loss and these tests do not allow diagnosis, then exploratory surgery is the next step. However, it is very easy to not be able to find the cause of the bleeding in these patients.

Medical management

If the patient is not exsanguinating, the cause is known or strongly suspected, and the patient has not had 5-7 days of appropriate medical therapy, then medical therapy is often reasonable as opposed to doing a major diagnostic work up. In distinction, if the patient is exsanguinating or if the patient has not shown any appreciable response to 5-7 days of appropriate medical therapy for the ulceration, then it is usually reasonable to surgically resect the ulcerated area. Note – when I say “response”, I am not referring to the patient being cured; I am referring to clear evidence of improvement. If surgery will be considered, it is usually very wise to perform gastroduodenoscopy before the surgery to be sure that you find all of the sites of ulceration. It is very easy to fail to detect an ulcer at surgery, and endoscopy usually allows one to easily find all areas of ulceration. Sometimes intraoperative endoscopy is necessary to help the surgeon find the ulcer(s).

If medical management is elected, first be sure to remove the cause of the GUE. If the cause is not removed, medical management tends to be far less successful. Next, be sure that the patient is well hydrated; healing of the gut requires or is at least benefitted by adequate perfusion. If there is significant gastroduodenal reflux of bile, metoclopramide or cisapride may be helpful in preventing bile from entering and/or staying in the stomach and augmenting the ulcerogenic process.

**H-2 receptor antagonists** are commonly used. Cimetidine, ranitidine, and famotidine are good medications for decreasing the gastric hydrogen ion concentration. Famotidine (0.5 mg/kg) only needs to be given once or twice daily. The primary value of the H-2 receptor antagonists is in treating existing ulcers and erosions. They can be helpful in preventing some types of ulcers, but they are not effective in preventing ulcers due to NSAIDs or due to steroids.

**Proton pump inhibitors** (i.e., omeprazole, lanosprazole, pantoprazole) are the most effective inhibitors of gastric acid secretion we currently have available. The dose of omeprazole is 0.7-1.5 mg/kg qd, although I have often used it at up to 2 mg/kg bid in patients with severe reflux esophagitis or gastrinomas. The dose of lanosprazole (Previcid) and pantoprazole (Protonix) is 1 mg/kg IV (not approved for use in dogs). It generally takes 2-5 days for a PPI to have maximal efficacy.
**Misoprostol** (Cytotec®) is a prostaglandin E analog which was primarily designed to be a prophylactic drug used to prevent GUE due to NSAIDs. It is typically used at a dose of 2-5 µg/kg, 3-4 times daily. It is the best drug available that can be used to prevent NSAID-induced ulceration, but it is not uniformly effective in dogs.

**Sucralfate** seems to be extremely effective in protecting those areas which are already ulcerated and helping them heal. The only common side-effect is constipation.

**Selected readings:**


