

An approach to gastrointestinal haemorrhage

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Gastrointestinal (GI) haemorrhage is an important cause of blood loss. Haematemesis and melaena are suggestive of GI haemorrhage, but these signs are not always noted on initial assessment and even acute severe GI haemorrhage may be overlooked if signs localising blood loss to the GI tract are not present.^{1,2} GI haemorrhage tends to be reported more frequently in dogs than in cats. In both canine and feline patients, GI ulceration is the most common cause of GI haemorrhage,^{2,5} so it is reasonable to administer GI protectants prior to confirming the cause of GI haemorrhage. In dogs, non-steroidal anti-inflammatory drugs (NSAIDs) and hepatic disease are frequent causes of GI ulceration.³ In cats, neoplasia is a common cause of GI ulceration.² Severe thrombocytopenia should also be considered as a potential cause of GI haemorrhage in dogs.⁶ GI haemorrhage may be acute or chronic, occult (no visible blood) or overt (grossly visible blood), and can vary from mild self-limiting cases to severe life-threatening conditions.⁶ Mild cases may progress to become life-threatening, so it is important to rapidly identify patients with GI haemorrhage and to initiate therapies to prevent deterioration. With acute severe GI haemorrhage, the primary objective is to rapidly assess the patient's cardiovascular status and institute aggressive resuscitative efforts if shock is present. Most cases of GI haemorrhage respond well to medical management, however surgery may occasionally be indicated.

AETIOLOGY

GI haemorrhage may originate in the oesophagus, stomach, small intestine or colon. Pathologies associated with GI haemorrhage can be divided into three broad categories: diseases causing GI ulceration, coagulopathies and vascular anomalies.^{3,6}

In both dogs and cats, the most commonly reported cause of GI haemorrhage is GI ulceration (Table 1).^{2,5} The severity of haemorrhage associated with GI ulcers depends on the extent of mucosal erosion and whether there is erosion into underlying mucosal arteries.⁷ Diseases associated with GI ulceration in dogs and cats are listed in Table 1. NSAIDs and hepatic disease are the most commonly reported risk factors for GI ulcers in dogs.³ Neoplasia is a common risk factor for GI ulcers in cats, particularly systemic mastocytosis, gastrinoma and intestinal lymphosarcoma.³ Inflammatory bowel disease is also an important non-neoplastic cause of GI ulceration in dogs and cats.² The incidence of stress-induced ulcers in dogs and cats has not been determined; however, stress ulcers have been reported in some animals following surgery or hypovolaemic events and should be considered in patients that develop GI haemorrhage while hospitalised.^{2,8} Coagulopathies are another important cause of GI haemorrhage. Thrombocytopenia is the most common coagulopathy resulting in GI haemorrhage in dogs.⁶ Other coagulation disorders associated with GI haemorrhage



Figure 1: Haematemesis in a five-year-old male neutered Saint Bernard receiving NSAIDs for hip dysplasia.



Figure 2: A mixture of normal stool and melaena (black section of stool in the middle of the image) from the same dog shown in Figure 1.



Figure 3: Haematochezia in an 11-year-old mixed breed dog with a small intestinal tumour at the ileo-colic junction.

include rodenticide toxicity, disseminated intravascular coagulation (DIC), and coagulation factor deficiencies.^{4,6} Vascular anomalies are a common cause of GI haemorrhage in humans. However, only a small number of cases of vascular anomaly have been reported in the veterinary literature and it appears to be an uncommon cause of GI haemorrhage in dogs and cats.⁹

HISTORY AND PHYSICAL EXAMINATION

Patients with extensive GI haemorrhage, vomiting, diarrhoea or ulcer perforation may present in a state of shock due to anaemia, hypovolaemia, endotoxaemia or sepsis. Examination findings consistent with shock include tachycardia, weak or thready arterial pulses, cool extremities, prolonged capillary refill time, and pale mucous membranes. Aggressive resuscitative therapy with administration of IV fluids is the first priority to reverse the state of shock. Localisation of the site of GI haemorrhage and tailored therapies may need to be delayed until the cardiovascular system has been stabilised.

Once resuscitation has been initiated, a complete history should be taken and a thorough physical exam should be performed. In patients with GI haemorrhage, a history of aspirin or other NSAID administration is not uncommon.^{3,10,11} There are case reports of GI ulceration, haemorrhage and perforation in veterinary patients that have received NSAIDs even at recommended therapeutic doses.¹⁰ A history of haematemesis (vomiting of frank blood or dark material like coffee grounds), haematochezia (passage of frank blood) or melaena (black tarry stool) suggests the GI tract as a source of haemorrhage (Figures 1, 2 and 3). However, these signs are not always evident and may not appear until significant GI haemorrhage has occurred.^{2,3,10} Note that diseases of the nasal cavity and oropharynx can occasionally cause haematemesis and melaena due to swallowing blood from epistaxis or haemoptysis. Also note that activated charcoal, bismuth and diets high in iron can result in dark stools which should not be confused with melaena.¹² In cases of thrombocytopenia or other coagulopathies, there may be a history of bleeding from other sites of the body including the nasal cavities or urinary tract.

The abdomen should be examined carefully in any patients with suspected GI haemorrhage. Abdominal palpation may

Table 1: Diseases associated with gastrointestinal ulceration in dogs and cats.

Drug administration
NSAIDs
Corticosteroids
Mucosal trauma
Foreign bodies
Iatrogenic (endoscopy)
Systemic and metabolic diseases
Hepatic disease
Uremia
Pancreatitis
Hypoadrenocorticism
Neoplasia
Systemic mastocytosis
Gastrinoma
GI neoplasia (lymphoma, adenocarcinoma, leiomyoma, leiomyosarcoma, haemangioma)
Ischaemic events
Gastric dilatation and volvulus (GDV)
Mesenteric volvulus
Mesenteric thrombosis
Intussusception
Infectious disease
Fungal (<i>Pythium</i> , <i>Histoplasma</i>)
Bacterial (<i>Salmonella</i> , <i>Clostridium</i> spp, <i>Campylobacter</i>)
Parasitic (hookworms, whipworms, roundworms, <i>Coccidia</i>)
Viral (parvovirus, coronavirus)
Algal (<i>Protothecosis</i>)
Neurological disease
Head trauma
Intervertebral disc disease (IVDD)
Stress of critical illness
Major surgery
Hypovolaemia
Sepsis
Miscellaneous
Inflammatory bowel disease (IBD)
Haemorrhagic gastroenteritis (HGE)
Polyps
Vascular anomalies

localise areas of pain, induce nausea, identify masses or foreign objects, or detect abdominal distention or a fluid wave. Splenomegaly or hepatomegaly may be identified in patients with neoplasia or hepatic diseases. A rectal examination should be performed to detect frank blood or melaena and to look for masses or foreign bodies. Examination of the mucosal, skin surfaces and sclera may reveal petechiae in severely thrombocytopenic patients (Figures 4a, 4b and 4c). A search for subcutaneous nodules or masses may detect mast cell tumours. Localisation of the site of GI haemorrhage is important because the aetiologies, diagnostic tests and therapies for upper versus lower GI haemorrhage may differ and because upper GI haemorrhage tends to be more severe (although haemorrhage from any site in the GI tract can be serious).^{4,12,13} Haematemesis or melaena typically suggests upper GI haemorrhage.¹² However, the colour of the blood in the stool or vomitus depends on the amount of time the blood remains in the GI tract and not necessarily the site of bleeding, so delayed GI transit time and retention



Figure 4a, 4b, 4c: Petechia and ecchymosis are often noted on the mucous membranes (4a), ventral abdomen (4b) and sclera (4c) in dogs with marked thrombocytopenia.

of blood in the colon could result in melaena associated with a lower GI tract lesion.^{11,12,14} Haematochezia is usually reflective of large intestinal, rectal or anal haemorrhage. However, severe acute upper intestinal haemorrhage can act as a cathartic, significantly decreasing GI transit time and resulting in frank blood in the stool.^{11,13}

DIAGNOSTIC TESTS

Patients with symptoms of shock should have emergency minimum blood tests (haematocrit, total solids, blood urea nitrogen [BUN], blood glucose and, if available, lactate, pH and electrolytes) while resuscitative efforts are initiated. Once resuscitation has commenced or the patient's condition has stabilised, other diagnostic modalities should be considered to confirm GI haemorrhage and localise the source of bleeding within the GI tract.

Tests to detect the presence of gastrointestinal haemorrhage

Even if GI haemorrhage is not obvious based on the history or physical exam, anaemia of undetermined origin should prompt consideration of GI blood loss. Microcytic hypochromic anaemia (iron deficiency anaemia) is reported following chronic GI blood loss, however iron deficiency anaemia takes time to develop so normocytic normochromic anaemia is more common in cases of recent GI haemorrhage.^{3,6} An elevated haematocrit and a relatively normal plasma protein concentration in a patient with acute haemorrhagic diarrhoea is suggestive of haemorrhagic gastroenteritis.¹⁵ A high BUN-to-creatinine ratio (>20 using conventional [US] units) has been reported with GI haemorrhage, particularly in cases of upper GI haemorrhage.¹⁴ However, many patients with GI haemorrhage do not have an elevation in BUN concentration.^{6,14}

In equivocal cases of GI haemorrhage, a faecal occult blood test can be performed. False positive results may occur due to dietary factors (red meat, fish, fruits, vegetables) or certain types of bacteria within the GI tract.¹⁶ It has been recommended that patients be fed a meat-free diet for at least 72 hours before a faecal occult blood test.¹⁵ Although causes of false positive results must be considered, a negative faecal occult blood test definitively rules out significant GI haemorrhage.¹

Tests to identify underlying causes

Once GI haemorrhage is confirmed or suspected, attempts should be made to identify an underlying cause. This often includes a coagulation profile, complete blood count (CBC), biochemistry profile, electrolytes, adrenocorticotrophic hormone (ACTH) stimulation testing, imaging and endoscopy as indicated.

The coagulation profile may identify coagulopathies (rodenticide toxicity or clotting factor deficiencies), and may also help detect coagulation defects that are not the primary cause of GI haemorrhage but that significantly exacerbate blood loss. It is very important to evaluate platelet count because immune-mediated

thrombocytopenia (ITP) is a common cause of GI haemorrhage in dogs.⁶

Hepatic and renal diseases are reported causes of GI ulceration, so particular attention should be paid to biochemical markers reflective of these diseases (alkaline phosphatase, alanine aminotransferase, aspartate aminotransferase and bilirubin for hepatic disease; urea, creatinine, and phosphorus for renal disease). Hypoadrenocorticism can be associated with severe GI haemorrhage in dogs, so electrolyte levels should be evaluated and an ACTH stimulation test should be performed if hypoadrenocorticism is suspected or if no other cause of GI haemorrhage is identified.¹⁷ Faecal smears, cultures and parvovirus testing may be indicated if infectious disease is suspected.

Radiographs may detect foreign bodies, masses, or free air in the peritoneal cavity (which suggests GI perforation). Ultrasonography can also be used to identify foreign bodies and masses, and may help detect GI perforation.^{18,19} Ultrasonography can even be used to detect mucosal ulceration in dogs and cats, through evaluation of intestinal wall structure and thickness to look for defects or craters (Figure 5).¹⁹ When used serially, ultrasonography may help to determine mucosal changes in response to therapy and has suggested the need for surgery in some instances.¹⁹ Endoscopy is considered the most sensitive test to evaluate upper GI tract ulceration, although patients must be optimally resuscitated before the procedure.^{7,20} Endoscopy often provides a definitive diagnosis, helps to determine prognosis, and may have therapeutic benefits (such as foreign body removal). In addition to allowing direct visualisation of the mucosa, endoscopy allows collection of biopsies for histology and culture, which may be required to diagnose neoplastic or inflammatory lesions and infectious diseases. The disadvantages of endoscopy include the need for anaesthesia, restriction to the proximal GI tract and colon, potential to exacerbate GI haemorrhage, possibility of causing iatrogenic ulcer perforation, and possibility of creating marked pneumoperitoneum in cases of pre-existing gastrointestinal perforation.¹¹

THE THERAPY

The treatment priority in patients with GI haemorrhage is to stabilise the cardiovascular system, control ongoing haemorrhage, manage existing ulcers, prevent bacterial translocation, and identify and treat the underlying cause.

Blood transfusions

Depending on the duration and extent of blood loss, a transfusion of packed red blood cells, whole blood or oxyglobin may be indicated. In patients with severe acute GI haemorrhage, blood transfusion is often part of the initial resuscitation protocol (Figure 6). In patients that do not initially display signs of shock, determining when a blood transfusion should be given is not clearly defined and depends on the rate and volume of blood loss, haemodynamic status, initial and serial haematocrits, and concurrent illness.²¹ If the patient develops clinical signs

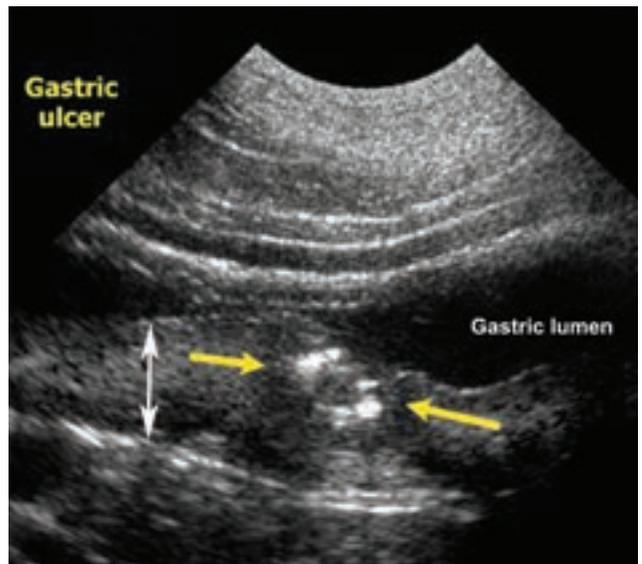


Figure 5: Sonographic image of the stomach depicting a hyperechoic outlined circular erosion (yellow arrows) arising from the gastric mucosa. Also note the marked gastric wall thickening (white arrow) and fluid filled lumen.

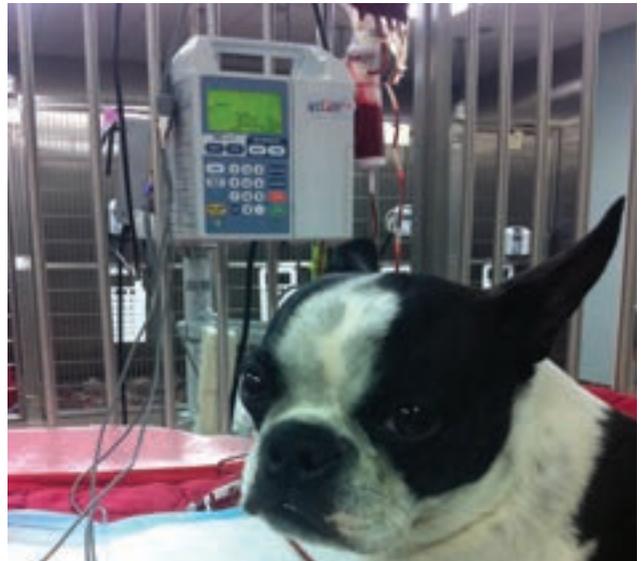


Figure 6: A four-year-old female Boston Terrier receiving a fresh whole blood transfusion for treatment of marked anaemia secondary to gastrointestinal haemorrhage.

attributable to decreased oxygen delivery (tachycardia, hyperlactataemia, tachypnoea), or if serial measurements reveal a decreasing haematocrit after initiating therapy, a blood transfusion may be indicated.²¹

Ulcerative gastrointestinal haemorrhage

Animals with haematemesis and melaena should be treated presumptively for GI ulcers until proven otherwise. Medications known to cause ulcers (NSAIDs and steroids) should be discontinued, unless they are considered essential to therapy. It is reasonable to administer GI protectants before confirming the cause of GI haemorrhage, because ulcers are the most common cause of GI haemorrhage in dogs and cats and these medications have a wide safety margin. Commonly used

GI protectants include: gastric acid suppressants such as histamine-2 receptor antagonists (cimetidine, ranitidine, famotidine) and proton pump inhibitors (omeprazole, pantoprazole), mucosal binding agents (sucralfate) and synthetic prostaglandins (misoprostol). There are no veterinary studies to conclude which combination of gastroprotectants is most effective in the management of GI ulcers. However, one study demonstrated that famotidine (0.5mg/kg IV q12hr), omeprazole (1mg/kg orally q24hr) and pantoprazole (1mg/kg IV q24hr) each significantly suppressed gastric acid secretion in dogs, while ranitidine (2mg/kg IV q12hr) failed to show significant gastric acid suppression at the dose evaluated.²² Most cases of suspected GI ulceration are managed with sucralfate and either a histamine-2 antagonist or proton pump inhibitor. In cases of NSAID toxicity, misoprostol may provide additional benefit. Consideration should be given to the route of drug administration because absorption of oral medications may be limited in critically ill patients and many dogs with GI haemorrhage are vomiting. In patients with persistent vomiting, metoclopramide (constant rate IV infusion 1-2mg/kg/24hr) is often administered initially. Cases refractory to metoclopramide may benefit from additional antiemetics such as ondansetron. Many causes of GI haemorrhage are associated with pain, so analgesics (such as opioids) should be considered.

In cases with significant GI haemorrhage, broad spectrum antibiotics are warranted due to risk of GI mucosal barrier compromise and bacterial translocation. Common broad spectrum antibiotic combinations include: a penicillin with an aminoglycoside or fluoroquinolone, or a cephalosporin with metronidazole and an aminoglycoside or fluoroquinolone. Ideally, samples for culture and sensitivity (urine and blood) should be collected before starting antibiotic therapy.

Most cases of GI haemorrhage due to GI ulceration can be managed medically. However, surgery may be indicated for pre-existing surgical disease (foreign body, tumour, septic abdomen), for patients at risk of exsanguination or perforation (based on endoscopy or serial sonographic evaluation), or if the patient fails to respond to medical therapy.

Because of the wide range of disease conditions that can result in GI ulceration and haemorrhage, therapy directed toward correcting the underlying cause is variable (such as surgery for foreign bodies or tumours, steroids for hypoadrenocorticism, immunosuppressives for ITP, or discontinuation of NSAIDs). While managing the underlying causes of GI haemorrhage, it is also important to consider related or unrelated coagulation abnormalities (such as liver disease causing ulceration and a clotting factor deficiency) and to address concurrent diseases that may exacerbate GI haemorrhage (such as uraemia in a patient on NSAIDs).

Thrombocytopenia

Glucocorticoids remain the standard-of-care for treating

primary or secondary ITP. Glucocorticoids should be administered at standard immunosuppressive doses. There are limited data to promote one protocol over another. In the authors' experience, dexamethasone (0.25-0.5mg/kg q24h) is usually given to animals with an IV catheter, and prednisone (2-4mg/kg q24h) is used for oral treatment (with a minimum initial treatment period of two weeks).

Cytotoxic immunosuppressive drugs may be considered if there is inadequate response to glucocorticoids. Azathioprine and cyclophosphamide are both potent immunosuppressive drugs, although case studies have shown more improvement and better outcomes with azathioprine. Azathioprine (2mg/kg PO q24h) is tapered in a similar fashion to prednisone. A potential side-effect of cytotoxic drugs is myelosuppression, which reduces regenerative erythropoiesis and thrombopoiesis. Dogs on azathioprine have variable sensitivity to myelosuppression, so CBCs should be monitored every one to two weeks to evaluate erythrocyte, neutrophil and platelet counts. Other cytotoxic immunosuppressive agents are generally reserved for dogs with refractory ITP or dogs not tolerating other drugs. These immunosuppressive agents include mycophenolate, leflunomide, and cyclosporine. Cyclosporine does not typically cause myelosuppression and has a rapid onset of action, but it has highly variable oral bioavailability. Treatment with cyclosporine should be started at 10mg/kg PO q12hr with a target blood level of 600ng/ml.

In patients with persistent GI haemorrhage as a result of thrombocytopenia, vincristine may increase platelet release from the bone marrow.²³ The mechanism of action of vincristine and its efficacy in cases of ITP is controversial, however one trial demonstrated benefit in dogs.²⁴ Vincristine is cheap, easy to administer, and has low risk of toxicity, so this drug is routinely given to ITP patients within the first 48 hours of admission at a dose of 0.02mg/kg (dogs <15kg) or 0.5mg/m² (dogs >15kg).

PROGNOSIS

Many cases of GI haemorrhage are self-limiting. The prognosis varies depending on the underlying cause. In cases of moderate-to-severe GI haemorrhage requiring a blood transfusion, the prognosis is reportedly fair to poor, with a mortality rate of 29-45% depending on underlying cause.⁶

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Reader Questions and Answers

1: RISK FACTORS FOR GI ULCERATION IN DOGS INCLUDE:

- A: Parvovirus, uremia, hyperadrenocorticism
- B: Inflammatory bowel disease, iron-rich diet, pancreatitis
- C: Intestinal foreign body, azathioprine, splenomegaly
- D: Hepatic disease, hypoadrenocorticism, NSAIDs
- E: Lipomas, mesenteric thrombosis, head trauma

2: WHAT IS THE MOST COMMON COAGULOPATHY CAUSING GI HAEMORRHAGE IN DOGS?

- A: Rodenticide toxicity
- B: Immune-mediated thrombocytopenia
- C: Factor XII deficiency
- D: Disseminated intravascular coagulation
- E: Von Willebrand disease

3: WHAT TYPE OF ANAEMIA IS MOST COMMON IN CASES OF RECENT GI HAEMORRHAGE?

- A: Normocytic, hypochromic
- B: Normocytic, normochromic
- C: Macrocytic, normochromic
- D: Microcytic, hypochromic
- E: Microcytic, normochromic

4: WHAT IS THE JUSTIFICATION FOR BROAD-SPECTRUM ANTIBIOTICS IN PATIENTS WITH SEVERE GI HAEMORRHAGE?

- A: Bacterial infection is the most common cause of GI haemorrhage
- B: Antibiotics help prevent GI perforation
- C: Risk of compromised GI mucosal barrier and bacterial translocation
- D: Reduce secondary infection following administration of immunosuppressive medication
- E: Antibiotic prophylaxis prior to surgery

5: REGARDING GI HAEMORRHAGE, THE COLOR OF BLOOD IN THE STOOL OR VOMITUS PRIMARILY DEPENDS UPON:

- A: Location of haemorrhage within the GI tract
- B: Underlying cause of GI haemorrhage
- C: Volume of blood lost through the GI tract
- D: Amount of protein in the diet
- E: Amount of time the blood remains in the GI tract

6: WHICH MEDICATIONS ARE COMMONLY USED GASTROPROTECTANTS?

- A: Sucralfate, omeprazole, famotidine
- B: Cyclosporine, sucralfate, aspirin
- C: Ranitidine, vincristine, bismuth
- D: Ondansetron, mycophenolate, famotidine
- E: Metoclopramide, pantoprazole, cimetidine

ANSWERS: 1: D, 2: B, 3: B, 4: C, 5: E, 6: A