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Chapter 130

GASTROINTESTINAL HEMORRHAGE

Søren R. Boysen, DVM, DACVECC

KEY POINTS

- Gastrointestinal (GI) hemorrhage is an important cause of blood loss anemia.
- In dogs and cats GI ulceration is the most commonly reported cause of GI hemorrhage.
- Nonsteroidal antiinflammatory drugs and hepatic disease are frequent causes of GI ulceration in dogs.
- Neoplasia is a common cause of GI ulceration in cats.
- Severe thrombocytopenia should not be overlooked as a cause of GI hemorrhage in dogs.
- Hematemesis and melena suggest GI hemorrhage but are not always noted.
- With acute severe GI hemorrhage, the primary objective is to rapidly assess the patient's cardiovascular status and institute aggressive resuscitative efforts if shock is present.
- It is reasonable to administer GI protectants before confirming the cause of GI hemorrhage.
- Most cases of GI hemorrhage respond well to medical treatment, although surgery may be indicated in others.

INTRODUCTION

Gastrointestinal (GI) hemorrhage is an important cause of blood loss anemia and a potentially life-threatening condition in dogs.[1] It is reported less frequently in cats. It may be acute or chronic, occult (no visible blood) or overt (grossly visible blood), and can vary from mild, self-limiting states to severe life-threatening conditions. Significant GI hemorrhage can often be detected during history and physical examination. However, on occasion even acute severe GI hemorrhage may be overlooked if signs localizing blood loss to the GI tract are not present or if concurrent disease obscures the diagnosis.[2,3] In addition, because even mild cases may progress to life-threatening events, it is important to rapidly identify patients with GI hemorrhage and institute therapies to prevent their deterioration.

ETIOLOGY

GI hemorrhage in dogs and cats can be the result of a primary insult to the GI tract or may be secondary to a systemic disease process. It may originate in the esophagus, stomach, small intestine, or large intestine. As such, a number of pathologic processes have been associated with GI hemorrhage. In general, these can be divided into three broad categories: diseases causing ulcers, diseases causing coagulopathies, and diseases associated with vascular anomalies. Animals may have single or multiple predisposing causes.[1,4]

The most commonly reported cause of GI hemorrhage in dogs and cats is GI ulceration.[3-6] The severity of GI hemorrhage associated with ulcers varies with the degree and extent of mucosal erosion. With erosion into an underlying artery, the magnitude of bleeding is related to the size of the arterial defect and the diameter of the artery.[7] Diseases associated with GI ulceration in dogs and cats are listed in Box 130-1. Nonsteroidal antiinflammatory drugs (NSAIDs) and hepatic disease are the most commonly reported risk factors for ulcers in dogs (Color Plate 130-1).[4] Neoplasia is a common risk factor for ulcers in cats, with systemic mastocytosis, gastrinoma, intestinal lymphosarcoma, and adenocarcinoma being the most commonly reported tumors.[3] Inflammatory bowel disease may also be an important nonneoplastic cause of GI ulceration in cats and dogs.[3] Stress ulcers are a frequent cause of GI hemorrhage in critically ill human patients and have been reported in dogs and cats following hypovolemia and surgery.[3,8] The true incidence and significance of stress ulcers in critically ill cats and dogs has not been determined, but should be considered in patients that develop GI hemorrhage while in the hospital.

Coagulation disorders associated with GI hemorrhage include rodenticide toxicity, disseminated intravascular coagulation, coagulation factor deficiencies (factor XII and prekallikrein deficiency), and thrombocytopenia.[1,5] Thrombocytopenia is the most common coagulation disorder resulting in GI hemorrhage in dogs and should not be overlooked.[1] Coagulation disorders resulting in GI hemorrhage appear to be less common in cats.

Vascular anomalies, because of the high incidence of varices, are a common cause of GI hemorrhage in humans. In contrast, only a few cases of vascular anomaly have been reported in the veterinary literature and it appears to be an infrequent cause of GI hemorrhage in dogs and cats.[9] It should be considered when more common causes of GI hemorrhage have been ruled out.

HISTORY AND PHYSICAL EXAMINATION

With extensive hemorrhage, vomiting, diarrhea, or ulcer perforation, patients with GI hemorrhage may present in a state of shock due to blood loss, hypovolemia, endotoxemia, or sepsis. Examination findings consistent with shock include tachycardia, diminished or thready arterial pulses (particularly peripheral), cool extremities, prolonged capillary refill time, and pale mucous membranes. Aggressive resuscitative therapies to reverse the state of shock take precedence (see Chapters 10 and 65, Shock and Shock Fluids and Fluid Challenge, respectively), and localization of the site of hemorrhage and tailored therapies may need to be delayed until the cardiovascular system is stable.

Once resuscitative efforts have commenced, a complete history and physical examination should be performed. Hematemesis (vomitus with the appearance of coffee grounds or

frank blood), hematochezia (passage of bright red or frank blood with or without stool), or melena (black tarry stool) suggests the GI tract as a source of hemorrhage. However, these signs are not always evident clinically and may not appear until significant GI hemorrhage has occurred.[3,4,10] With duodenal hemorrhage, if reflux of duodenal contents into the stomach is insufficient, blood may not be visible in the vomitus.[11] However, when it is present, hematemesis suggests ongoing blood loss.[12] Diseases of the nasal cavity and oropharynx occasionally can cause hematemesis and melena from swallowing blood of epistaxis or hemoptysis (coughing of blood), and these causes should be considered. In addition, activated charcoal, bismuth (Pepto-Bismol), and diets high in iron can result in dark stools and should not be confused with melena.[13]

A history of aspirin or other NSAID administration is not uncommon.[4,10,14] There are case reports of GI ulceration, hemorrhage, and GI perforation occurring in veterinary patients that have received selective cyclooxygenase inhibitors at recommended therapeutic dosages.[10] Decrease or loss of appetite with or without other signs of GI disease should prompt consideration of GI side effects in any patients receiving NSAIDs. The medication should be discontinued and the patient should be examined. In cases of thrombocytopenia or coagulation disorders, there may be a history of bleeding from other sites of the body including the nasal cavities or urinary tract. Thorough examination of the mucosal surfaces may reveal petechiae in severely thrombocytopenic patients. A search for subcutaneous nodules or masses may detect underlying mast cell tumors.

Because GI hemorrhage may be insidious in onset, especially when chronic, the abdomen should be examined carefully. Abdominal palpation may localize areas of pain (tenderness, voluntary or involuntary guarding) or induce nausea, identify masses or foreign objects, or detect abdominal distention or a fluid wave. Splenomegaly or hepatomegaly may be identified in patients with mastocytosis, other neoplasia, or hepatic diseases. During initial evaluation or resuscitation of the patient, a careful rectal examination should be performed to detect frank blood or melena and to look for masses or foreign bodies.

Although hemorrhage from any site in the GI tract can be serious, upper GI hemorrhage tends to be more severe.[12,13] In addition, the etiology as well as the diagnostic tests and therapies for upper and lower GI hemorrhage may vary, making localization of the site of hemorrhage important.[5,13] Hematemesis or melena suggests upper GI hemorrhage.[13] However, it is important to remember that it is the amount of time the blood remains in the GI tract and not necessarily the site of bleeding that determines its color.[13,14] Delayed GI transit time and retention of blood in the colon could result in melena associated with a lower GI tract lesion.[13,15] Hematochezia is usually reflective of large intestinal, rectal, or anal hemorrhage; however, severe acute intestinal hemorrhage can act as a cathartic, significantly decreasing GI transit time.[12-14] This may result in the passage of frank blood in the stool following significant blood loss into the upper GI tract.[12,14]

DIAGNOSTIC TESTS

GI hemorrhage is confirmed when a source of bleeding is localized to the GI tract. Patients with signs of shock should have emergency minimum blood tests performed (hematocrit, total solids, blood urea nitrogen [BUN], glucose

Box 130-1 Diseases Associated With GI Ulceration and Hemorrhage in Dogs and Cats

Drug Administration

NSAIDs
Glucocorticoids

Systemic and Metabolic Diseases

Hepatic disease
Uremia
Pancreatitis
Hypoadrenocorticism

Ischemic Events

GDV
Mesenteric volvulus
Mesenteric thrombosis
Intussusception

Neurologic Disease

Head trauma
IVDD
Mucosal trauma
Foreign bodies

Fungal Infections

Pythium
Histoplasma

Bacterial Infections

Salmonella
Clostridium spp
Campylobacter
Helicobacter (controversial)

Parasitic Infections

Hookworms
Whipworms
Coccidia
Roundworms

Viral Infections

Parvovirus
Coronavirus

Algal Infections

Protothecosis
Systemic neoplasia
Mastocytosis
Gastrinoma

Gastrointestinal Neoplasia

Lymphoma
Adenocarcinoma
Leiomyoma
Leiomyosarcoma

Stress of Critical Illness

Major surgery
Hypovolemia
Sepsis

Miscellaneous

IBD
Polyps
HGE

GDV, Gastric dilatation-volvulus; *GI,* gastrointestinal; *HGE,* hemorrhagic gastroenteritis; *IBD,* inflammatory bowel disease; *IVDD,* intervertebral disk disease; *NSAIDs,* nonsteroidal antiinflammatory drugs.

and, if available, pH, lactate, and electrolytes) while resuscitative efforts and a search for the underlying cause are undertaken. In cases suspected to have hemoabdomen or septic peritonitis, abdominocentesis, emergency abdominal sonography, and diagnostic peritoneal lavage are warranted and may be performed during initial resuscitation of the patient. Once resuscitative efforts have commenced or the patient's condition has stabilized, other diagnostic modalities should be considered.

Tests to Help Detect Presence of Gastrointestinal Hemorrhage

When GI hemorrhage is not obvious on history or physical examination findings, certain hematologic and biochemical abnormalities may suggest its presence. Anemia of undetermined origin should prompt consideration of GI hemorrhage. The finding of microcytic, hypochromic anemia (iron deficiency anemia) is reported following chronic blood loss into the GI tract.[4] However, because iron deficiency anemia takes time to develop, normocytic normochromic anemia is more common in cases of recent GI hemorrhage.[1,4] A high BUN-to-creatinine ratio (>20) has been reported with GI hemorrhage, especially when it occurs in the upper GI tract.[15] This phenomenon has been explained by volume depletion and intestinal absorption of proteins, including digested blood, into the circulatory system.[15] Large bowel hemorrhage is reported to have little effect on BUN levels.[15,16] However, as diseases resulting in increased protein metabolism (fever, burns, infections, starvation, and administration of glucocorticoids) may also result in an increased BUN-to-creatinine ratio, they should be considered before concluding that GI hemorrhage is the cause.[1,15] It should also be noted that many dogs with GI hemorrhage do not have an elevation in the BUN concentration.[1]

In equivocal cases of GI hemorrhage a fecal occult blood test, most of which rely on the peroxidase activity of hemoglobin, may be performed. Although it may be helpful for detecting occult GI hemorrhage, diets containing red meat or having high peroxidase activity, such as fish, fruits, or vegetables, can cause false-positive results.[17] The presence of peroxidase-producing bacteria within the GI tract may also cause false-positive results.[17] These factors must be considered when interpreting positive chemical-based fecal occult blood test results.

It has been recommended that animals be fed a meat-free diet for at least 72 hours before a fecal occult blood test.[18] On the other hand, a negative fecal occult blood test result does rule out significant GI hemorrhage.[2] When significant gastric hemorrhage is suspected but not confirmed, passage of a nasogastric tube and aspiration of the stomach contents may confirm and help localize the site of GI hemorrhage, although the procedure may cause discomfort and false-negative results have been reported.[11,16]

Tests to Help Identify Underlying Causes

Once GI hemorrhage is confirmed or suspected, a search for an underlying cause should be pursued. This often includes a coagulation profile, complete blood count, routine biochemistry profile, electrolytes, adrenocorticotropic hormone stimulation testing, imaging, and endoscopy as indicated.

The coagulation profile may identify coagulopathies such as rodenticide intoxication or clotting factor deficiencies. It may also detect prolonged bleeding times that are not the direct cause of GI hemorrhage but that significantly exacerbate blood loss. The platelet count is important, because immune-mediated thrombocytopenia is a common cause of moderate to severe GI hemorrhage in dogs.[1] An elevated hematocrit in a patient with acute hemorrhagic diarrhea and a relatively normal plasma protein concentration is suggestive of hemorrhagic gastroenteritis.[18]

Given that hepatic and renal disease are reported causes of GI ulceration and hemorrhage, particular attention should be paid to the biochemical markers reflective of these diseases (alkaline phosphatase, alanine aminotransferase, aspartate aminotransferase, and bilirubin in cases of hepatic disease; and urea, creatinine, and phosphorus in cases of renal disease). Because hypoadrenocorticism has been reported as a cause of severe GI hemorrhage in the dog, electrolyte levels should be evaluated and an adrenocorticotropic hormone (ACTH) stimulation test performed if another cause for GI hemorrhage cannot be found.[19] Fecal smears, cultures, and parvovirus testing may be indicated if infectious disease is suspected. Measurement of gastrin levels is recommended in cases of recurrent GI ulceration and in cases that fail to respond to medical therapy.[4]

Radiographs may detect foreign bodies, masses, or free air in the peritoneal cavity. Pneumoperitoneum is suggestive of GI perforation in a patient that has not undergone recent abdominal surgery. Although contrast radiographs may identify mucosal defects as a cause of GI hemorrhage, they generally have been replaced by ultrasonography and endoscopy.[4,16] Ultrasonography often will identify foreign bodies and masses, and may help to identify concurrent GI perforation when present.[20,21] The use of ultrasonography to identify ulcers in dogs has been described. It allows evaluation of the intestinal wall structure and thickness and can detect the presence of a defect or crater.[21] When used serially, it may help determine changes in response to therapy and has suggested the need for surgery in some instances.[21] Ultrasonography has also been reported in the assessment of cats with GI ulceration.[3]

Endoscopy is considered the most sensitive test to evaluate upper GI tract hemorrhage and ulcers, although patients must be optimally resuscitated before the procedure.[7,16] It often provides a diagnosis, helps assess prognosis, and may have therapeutic benefits (i.e., foreign body retrieval). In addition to allowing direct visualization of the mucosa, it permits biopsies for histology and culture, which may be required to identify lesions and infectious diseases (i.e., neoplasia, inflammatory bowel disease, protothecosis). The disadvantages of endoscopy include the need for anesthesia, its limitation to the proximal GI tract and colon, the potential to exacerbate GI hemorrhage, and the possibility of causing iatrogenic ulcer perforation.[14]

If the above diagnostic procedures fail to identify the cause of significant ongoing GI hemorrhage, abdominal exploratory surgery, scintigraphy using technetium-labeled red blood cells, and arteriography should be considered.[2,16,18] Scintigraphy has been demonstrated to aid in localization of GI hemorrhage in dogs, and arteriography may help identity GI vascular anomalies.[2,9,18]

TREATMENT

The treatment priority in patients with GI hemorrhage is to stabilize the cardiovascular system, control ongoing

hemorrhage, treat existing ulcers, prevent bacterial translocation, and to identify and address the underlying cause. The initial priority is to rapidly identify and reverse any signs of shock (see Chapters 10 and 65, Shock and Shock Fluids and Fluid Challenge, respectively).

Depending on the duration and extent of blood loss, administration of packed red blood cells, whole blood, or oxyglobin may be indicated. In the patient with severe acute GI hemorrhage, this is often implemented as part of the initial resuscitation protocol. In patients that do not display initial signs of shock, determining when a blood transfusion should be given is less clearly defined. The decision to transfuse all patients at a specified hematocrit remains controversial. The hematocrit at which a patient requires a transfusion will vary depending on the degree and rate of blood loss, hemodynamic status, initial and subsequent hematocrits, presence of concurrent illness, and severity of clinical signs.[22] If the patient displays clinical signs attributable to a decrease in oxygen delivery (i.e., tachycardia, hyperlactatemia, tachypnea) or if serial measurements reveal a decreasing hematocrit after initiating therapy, a blood transfusion is indicated.[22]

If GI hemorrhage is the result of a primary coagulopathy or is exacerbated by a secondary coagulopathy (i.e., disseminated intravascular coagulation, hepatic failure, shock, or dilution with aggressive fluid therapy), fresh frozen plasma should be considered. In patients with persistent GI hemorrhage as a result of thrombocytopenia, vincristine may increase the release of platelets from the bone marrow, although the function of these platelets has been questioned.[23]

Iced saline gastric lavage has been suggested as a therapy to decrease GI hemorrhage[5,6]; however, the current consensus in the human literature is that it be avoided. Iced saline gastric lavage has not been proven to slow hemorrhage, is known to cause discomfort, and can rapidly lower core body temperature, which was demonstrated to prolong bleeding in an experimental canine study.[6,14]

Animals with hematemesis and melena should be treated for GI ulcers until proven otherwise. Medications known to cause ulcers should be discontinued (i.e., NSAIDS). Given the association between GI hemorrhage and steroids in dogs, unless they are considered essential to therapy (i.e., hypoadrenocorticism, immune-mediated diseases), they should also be discontinued.

It is reasonable to administer GI protectants before confirming the cause of GI hemorrhage, given that ulcers are the most common cause of GI hemorrhage in dogs and cats, and GI protectants have a wide safety margin. In addition, intraluminal gastric acid neutralization may slow GI hemorrhage by promoting mucosal homeostasis.[7,24] Commonly used GI protectants include acid suppressants such as histamine-2 receptor antagonists (cimetidine, ranitidine, famotidine) and proton pump inhibitors (omeprazole, pantoprazole), mucosal binding agents such as sucralfate, and synthetic prostaglandins such as misoprostol. There are no veterinary studies to conclude which gastroprotectants or combination of gastroprotectants are most efficacious in the management of GI ulcers. However, a study demonstrated that famotidine (0.5 mg/kg IV q12h), omeprazole (1 mg/kg PO q24h), and pantoprazole (1 mg/kg IV q24h) significantly suppressed gastric acid secretion in dogs, but ranitidine (2 mg/kg IV q24h) failed to show significant gastric acid suppression at the dosage evaluated.[24]

Although histamine-2 antagonists, proton pump inhibitors, sucralfate, and misoprostol have been administered concurrently, most cases of suspected GI ulceration are managed with either a histamine-2 antagonist or proton pump inhibitor and sucralfate.[4,14] In cases of NSAID toxicity, misoprostol may provide additional benefit. In deciding which medications to use, consideration should be given to the route of drug administration because absorption of medications administered orally in critically ill patients has been questioned, and many dogs with GI hemorrhage are vomiting, which may further limit the utility of oral medications. In patients that have persistent vomiting, antiemetics can be used. Metoclopramide, given as a constant intravenous infusion (1 to 2 mg/kg q24h), is often tried initially. Cases refractory to metoclopramide may benefit from additional antiemetics such as odansetron. Because many causes of GI hemorrhage are associated with discomfort and pain, analgesics such as an opioid should be considered.

In cases with significant GI hemorrhage, broad-spectrum antibiotics (i.e., a penicillin and an aminoglycoside or fluoroquinolone, or a combination of a cephalosporin, metronidazole, and an aminoglycoside or fluoroquinolone) are warranted because of the risk of GI mucosal barrier compromise and bacterial translocation. Ideally, samples for culture and sensitivity (i.e., urine and blood) should be collected before starting antibiotic therapy.

Most cases of GI hemorrhage can be managed medically. In cases of severe GI ulceration and hemorrhage refractory to medical treatment, endoscopic hemostasis may be beneficial. Ulcer hemostasis has been described by injecting epinephrine or 98% alcohol through an endoscope sclerotomy needle into the base of an ulcer.[7,25] The minimally invasive use of Endoclips or endoscopic thermal, electric, and laser cautery has been described to control GI hemorrhage secondary to vascular anomalies and ulcers in humans and may be applicable to veterinary medicine.[7,11] Surgery can be avoided in most cases, but is indicated for preexisting surgical disease (foreign body, tumor, septic abdomen) in 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 large number of disease conditions that can result in GI hemorrhage, therapy directed toward correcting the underlying cause is variable (i.e., surgery for foreign bodies or tumors, steroids for hypoadrenocorticism, immunosuppressives for immune-mediated thrombocytopenia, discontinuation of NSAIDs). In considering the underlying cause, it is important to consider related or unrelated coagulation abnormalities (i.e., liver disease causing ulceration and a clotting factor deficiency) and to address concurrent diseases that may exacerbate GI hemorrhage (i.e., uremia in a patient on NSAIDs).

PROGNOSIS

Many cases of GI hemorrhage are self-limiting and the prognosis varies with the underlying cause. In cases of moderate to severe GI hemorrhage requiring a blood transfusion, the prognosis is reportedly fair to poor, with a mortality rate of 29% to 45%.[1]

SUGGESTED FURTHER READING*

Liptak JM, Hunt GB, Barrs VRD, et al: Gastroduodenal ulceration in cats: Eight cases and a review of the literature, *J Feline Med Surg* 4:27-42, 2002.
A small study with a good review of the literature concerning GI ulcers in cats.
Stanton ME, Ronald BM: Gastroduodenal ulceration in dogs: retrospective study of 43 cases and literature review, *J Vet Intern Med* 3:238, 1989.
A nice retrospective study and review of GI ulceration in dogs, including the pathophysiology of ulcer development in dogs with various underlying diseases.
Waldrop JE, Rozanski EA, Freeman LM, et al: Packed red blood cell transfusions in dogs with gastrointestinal hemorrhage: 55 cases (1999-2001), *J Am Anim Hosp Assoc* 39:523, 2003.
One of the few publications investigating causes of GI hemorrhage in veterinary patients and an excellent paper addressing causes and management of patients with severe acute GI hemorrhage.
Washabau RJ: Acute gastrointestinal hemorrhage. Part I. Approach to patients, *Comp Cont Educ Pract Vet* 1:1317, 1996.
In conjunction with reference that follows, one of the most complete reviews of acute GI hemorrhage published in the veterinary literature.
Washabau RJ: Acute gastrointestinal hemorrhage. Part II. Causes and therapy, *Comp Cont Educ Pract Vet* 1:1327, 1996.
In conjunction with preceding reference, one of the most complete reviews of acute GI hemorrhage published in the veterinary literature.

*See the CD-ROM for a complete list of references.

Chapter 131
VOMITING AND REGURGITATION

Peter S. Chapman, BVetMed, DECVIM-CA, MRCVS

KEY POINTS

- It is important to differentiate between vomiting and regurgitation before proceeding with further diagnostic testing or therapy.
- Idiopathic megaesophagus is the most common cause of persistent regurgitation in the adult dog. Myasthenia gravis is the most common cause of secondary megaesophagus, accounting for 20% to 30% of all cases.
- Aspiration pneumonia is the most important cause of morbidity in the regurgitating patient.
- The multitude of differential diagnoses for vomiting can be subdivided into primary gastrointestinal and other causes.
- Abdominal radiographs should be obtained in all patients with acute vomiting. Abdominal ultrasonography may be a more useful imaging modality in patients with chronic vomiting.

DIFFERENTIATION OF VOMITING AND REGURGITATION

Before formulating a diagnostic and therapeutic plan, it is important to define the patient's clinical problem. Most importantly, vomiting and regurgitation must be distinguished; pet owners may not differentiate between the two problems, but the diagnostic investigations and treatment options will differ significantly. Occasionally pet owners will describe the harsh coughing and retching of canine infectious tracheobronchitis as vomiting. In most cases the problem can be defined accurately after taking a thorough history. Historic findings likely to assist in the differentiation between vomiting and regurgitation are presented in Table 131-1. Premonitory signs, active abdominal contractions, and bile are the characteristics that are most useful for making a diagnosis in vomiting animals and that are uncommonly seen in regurgitating patients. However, regurgitating animals may stretch and arch their necks, mimicking abdominal contractions, and the response to pain from an inflamed or ulcerated esophagus may resemble the classic signs of nausea.

It is important to distinguish true bile from the froth and saliva that animals with esophageal disease may regurgitate. Although relatively nonspecific, a further factor that may assist in the definition of the problem is the frequency of the episodes. Animals with esophageal disease may regurgitate saliva as frequently as hourly, yet remain bright and systemically healthy. A vomiting animal is unlikely to sustain this frequency of vomiting without becoming unwell.

REGURGITATION

Definition

Regurgitation is the passive ejection of food, water, or saliva associated with esophageal or, less commonly, pharyngeal disease.

Clinical Consequences of Regurgitation

The most significant clinical complication of regurgitation is aspiration pneumonia. Any patient with persistent regurgitation is at risk of aspiration pneumonia, and measures to reduce its occurrence should be instigated. Aspiration pneumonia is the most likely indication for hospitalization and intensive treatment of regurgitating patients. In the absence of aspiration pneumonia or other disease, most patients are able to maintain good hydration, although persistent regurgitation of undigested food may lead to marked weight loss.