

FROM POOP TO VOMIT: GASTROINTESTINAL DISEASES

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INTRODUCTION

The gastrointestinal tract (GI) consists of three main parts: stomach, small intestines and large intestines and has five main functions: motility, secretion, regulation, digestion and circulation. The digestive system is equally complicated, though it is mainly controlled by the CNS. Not only does it include the entire GI tract, but it also includes the oral cavity, pharynx, esophagus, salivary glands, pancreas, gall bladder and liver. It also is responsible for five main functions: ingestion, digestion, absorption, metabolism and excretion. There are many diseases which affect the GI tract only and those that affect both the GI and digestive tract.

STOMACH

The stomach is a very simple organ and is monogastric (only one chamber). It has three main functions: acts as a reservoir for food, breaks up and mixes food with gastric juices and begins the process of protein digestion. Food enters in from the oral cavity, past the pharynx, into the esophagus and eventually into the stomach. Food enters the stomach through the cardiac sphincter and leaves through the pyloric sphincter. The spleen is adhered to the greater curvature of the stomach.

SMALL INTESTINES

The small intestine is the major site of enzymatic digestion and absorption. Food enters through the pyloric sphincter into the small intestines. The small intestines are divided into three parts: duodenum, jejunum and ileum.

The duodenum is u-shaped with the pancreas lying on the bottom of the "U". The pancreatic duct (from the pancreas) and the common bile duct (from the gall bladder) open up into the duodenum. Within the duodenum is a series of glands which help to supply digestive juices. The duodenum leads into the jejunum. It is difficult to distinguish between the jejunum and ileum because they are a continuous long tube. The ileum eventually ends at the cecum.

The intestinal wall itself is made up of tiny leaf-shaped folds called villi. Inside each villus is a network of thin blood vessels which help to break down carbohydrates and proteins to the liver by way of the hepatic portal vein.

PANCREAS

The pancreas is a large extrinsic gland whose secretions are essential to the digestive process. It is classified as a mixed gland because it is both exocrine (secretes digestive enzymes and bicarbonate) and endocrine (produces hormones like insulin). The pancreas secretes digestive enzymes and bicarbonate into the duodenum via the pancreatic duct.

LARGE INTESTINES

The large intestines are structured similarly to the small intestines, but there are no villi and no digestive glands. Instead the large intestines are made up of more goblet cells which secrete mucus to help lubricate the feces as they pass through. The large intestine is made up into four parts: cecum, colon, rectum and anal sphincter. The cecum joins the ileum to the large

intestines. The colon is divided into three parts: ascending, transverse and descending. The rectum and anal sphincter mark the end of the large intestines.

LIVER & GALLBLADDER

The liver is the largest gland in the body. The liver is located in the cranial abdomen and is in direct contact with the diaphragm, stomach, duodenum and right kidney. The gallbladder is attached to the diaphragm on the ventral surface of the liver and lies between the main lobes. The liver is so complex that it contains a unique venous system called the hepatic portal system. The liver has many purposes including: carbohydrate, protein and fat metabolism, formation of bile, storage of vitamins and iron, detoxification of certain substances, regulation of body temperature and destruction of red blood cells.

The gallbladder's purpose is to store bile between meals, concentrate the bile by reabsorbing the water and then release it into the small intestines by way of the common bile duct. In certain disease processes the gallbladder may be removed. Without the gallbladder, bile is then discarded directly from the liver into the small intestines.

DISEASES OF THE STOMACH

ACUTE GASTRITIS: Acute gastritis is likely the most common cause of acute vomiting in dogs and cats and occurs from the inability of the mucosal barrier to protect itself. The literal meaning is: inflammation of the lining of the stomach. Sometimes vomiting will be severe causing losses of sodium, chloride and potassium. Most patients have limited changes in bloodwork. In the case of small animals (particularly neonates), blood glucose levels may decrease causing significant hypoglycemia.

The causes of acute gastritis are vast. Certain drugs or chemicals such as nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin, doxycycline, cephalosporins, lead or fertilizers can cause gastritis. No matter what the cause of the gastritis, a three-fold course of treatment, consisting of fluid therapy, dietary restriction and antiemetics (anti-vomiting), generally helps most animals recover in 24-48 hours.

The patient should be initially fasted for 12 hours to allow for a decrease in inflammation of the gastric mucosa. If the patient ceases to vomit during the 12 hours, then water and food trials should begin with a bland diet. Water should be initially given in small amounts because dogs, in particular, will drink excessively and cause themselves to vomit. Some animals will need to be coaxed to eat or tempted with a variety of food. Cats may not eat in a clinical setting due to being nervous, while most dogs tend to eat something. It is important to ensure your patient can tolerate food before discharging them to the owner.

GASTRIC DILATION AND VOLVULUS

Gastric dilation and volvulus (GDV), commonly known as bloat, is an acute life threatening condition, which is characterized by the malposition of the stomach when it rapidly fills with air and rotates. As the stomach fills with air, the vena cava becomes compressed, leading to a decrease in venous return from the heart. Depending on the degree of rotation of the stomach, a partial or complete blockage of the portal vein may also occur. This may cause the liver or pancreas to become ischemic (loss of bloody supply) to some degree. The spleen is located in the upper left region of the abdomen and is attached to the stomach via small blood vessels. When the stomach rotates, the spleen may also rotate and twist causing it too to become ischemic. Despite much research, the cause of GDV is unknown. However, there are risk

factors that have been identified. Most commonly GDV presents in dogs that are a large or giant breed and are deep chested (Great Dane, Saint Bernard, Irish Setter) and are middle age or older (mean age 7 years). Signs include non-productive writhing, abdominal distention and/or pain, anorexia and restlessness. Most patients will present in shock.

Treatment of the shock is the first priority. Because the patient is in shock and usually has severe abdominal pain, intravenous catheter placement, IV fluid therapy and pain medication should be started before diagnostics. Oxygen should also be administered because many GDV patients are in respiratory distress due to the enlarged size of the stomach pressing on the diaphragm. All vital and parameters should be monitored including pulse, respiration rate, blood pressure, mucous membrane color and ECG. Many GDV patients experience ventricular arrhythmias. A right lateral radiograph is the preferred diagnostic tool when ruling out GDV. A characteristic gas pattern is usually seen and is described as a “shelf” sign of tissues or a “boxing glove” of gas.

Once GDV has been diagnosed and therapy for shock has been initiated, gastric decompression should be performed followed by surgery. Surgery is always recommended because it is the only way to confirm whether the stomach is in correct anatomic position, that it is not necrotic or that the spleen is not thrombosed without it. During surgery, the stomach is repositioned and a permanent gastropexy is performed to prevent recurrence of GDV. The failure rate of gastropexy ranges between 3-8%.

No matter how stable a GDV patient was prior to and during surgery, all post surgery patients should be given a guarded prognosis. Post surgery all vitals need to be monitored as well as electrolyte and acid-base status. Fluid therapy should be tailored to the findings of the bloodwork and should not be discontinued until the patient is able to take in fluids orally.

DISEASES OF THE STOMACH & INTESTINES

ACUTE HEMORRHAGIC DIARRHEA SYNDROME /HEMORRHAGIC GASTROENTERITIS:

Acute hemorrhagic diarrhea syndrome (AHDS) is also known as hemorrhagic gastroenteritis (HGE) is primarily an intestinal disease rather than solely a gastric disorder even though vomiting is a primary symptom. AHDS/HGE is most commonly seen in the dog, with some rare reports of it being diagnosed in cats. AHDS /HGE is characterized by the acute loss of blood, fluids and electrolytes from the GI tract. Symptoms include acute vomiting (possibly hematemesis), anorexia and hematachezia. Animals tend to decline very quickly over 8-12 hours and present with moderate (7%) to severe (10-12%) dehydration. They may be in hypovolemic shock due to the severe amount of fluids lost over such a short amount of time. Bacteria and/or toxins may cross the gastric mucosal barrier and cause septic or endotoxic shock. The cause of AHDS /HGE is unknown, but certain breeds such as Yorkshire terriers, miniature schnauzers, miniature poodles or dachshunds may be at greater risk. Bloodwork typically reveals hemoconcentration, with a packed cell volume (PCV) being greater than 60% in the dog and greater than 50% in the cat. Therapy includes IV fluids, dietary restriction and antiemetic therapy. Generally these animals recover within 24-72 hours.

GASTRIC OR INTESTINAL OBSTRUCTION

Gastric or intestinal obstruction occurs from the inability of food to pass through part of the GI tract. There are many causes for obstruction of the GI tract, but more commonly it is because of a foreign object that the pet ingested or a mass. Animals generally present with symptoms that include abdominal pain, vomiting, anorexia, straining to defecate or diarrhea.

Once an obstruction has been diagnosed (through radiographs, ultrasound and/or contrast study) surgery is likely indicated. Though not common, sometimes a patient may present in shock. No matter whether the animal presents in shock or not, fluid administration and pain medication should begin immediately and be tapered to the patient's needs. Most of the time obstructed patients have some level of dehydration, which ideally should be corrected prior to surgery. The biggest concern with any gastric or intestinal obstruction is perforation. If a patient has a perforated stomach or GI tract, septic peritonitis (*see peritonitis section below*) becomes a primary concern. Depending on the type of foreign body the animal ingested, toxicities such as zinc or lead may be a concern as well. After surgery analgesia, antiemetic therapy, IV fluids and nutritional therapy should be administered.

ACUTE ABDOMEN/ABDOMINAL PAIN

When an animal presents with a sudden onset of severe abdominal discomfort or pain, it is known as an acute abdomen. Dogs are more commonly affected and the causes can be numerous. Often animals with acute abdominal signs are associated with life-threatening diseases. The abdominal pain is due to the stretching and inflammation of nerve fibers located within the organs and GI tract. Animals may present unwilling to walk or walking in a hunched appearance, collapsed, vomiting, having diarrhea or in shock. Due to the numerous diseases that can cause an acute abdomen, the discussion will be limited to differential diagnosis of the GI system. Differential diagnoses may include: infectious (parvo, panleukopenia), obstructive (GI foreign body, intussusception), gastric dilation with/without volvulus, ulceration, hemorrhagic gastroenteritis or perforation.

It is important to treat the emergent symptoms of the patient first. Most acute abdomen patients require immediate intravenous fluids. Those diseases that affect the GI tract can result in massive fluid losses, causing the animal to be in hypovolemic shock. Depending on the degree of shock and dehydration, crystalloids and/or colloids should be chosen. Pain should be addressed and appropriate analgesia should be administered. It is important to remember that many animals suffering from acute abdomens are extremely painful, and analgesia is required to help stabilize any painful animal in shock. Vitals, including blood pressure, should be constantly monitored.

SEPTIC PERITONITIS

Septic peritonitis is the inflammation of the peritoneum (the membrane which lines the abdominal cavity) due to the introduction of bacteria into the abdominal cavity from a perforation or rupture of a hollow viscus. It can occur from a necrotic tumor that has ruptured or perforated the intestines or, more commonly, from a GI foreign body that has penetrated the intestines. Perforating trauma (such as a stick or bullet) into the abdomen may also introduce bacteria causing peritonitis. The bacteria that enters the abdomen causes the inflammatory cascade to become activated. Depending on the cause, many patients may have limited symptoms such as anorexia or restlessness. As the disease progresses vomiting, diarrhea, abdominal pain and/or abdominal distention may be noted. Patients may present in shock with physical findings that include injected mucous membranes, dull mentation, fever, weak peripheral pulses and tachycardia. Generally, in advanced stages of peritonitis, the abdomen becomes very painful.

Treatment for septic peritonitis should be aggressive. Intravenous fluids (both colloids and crystalloids), antibiotics and analgesics should be started immediately. Antibiotics

should not be started until after an infected sample has been obtained. Plasma and heparin therapy may be necessary if DIC is suspected. Once the patient is considered stable, surgery is usually indicated to eliminate the contamination source. This may include removing a foreign body, a penetrating object, necrotic tissue, etc. The abdomen may be left open or closed, depending on the degree of infection and the cause of the peritonitis. Open abdominal drainage allows for septic material to drain out of the abdomen. It also allows for easy evaluations of the area and decreases the survival of anaerobic bacteria. Open abdomens create numerous intensive care challenges for technicians. Closed abdomens create less intensive care challenges for technicians. Closed suction drains are placed in the abdomen and the incision is closed around the drain. This technique does not allow for complete drainage of the abdomen, but the fluid that is removed is confined to the drain itself. The drain must be kept in place until the fluids amount are within physiologic limits and the fluid cytology shows no evidence of infection. Studies have shown survival between closed and open abdomens to be about the same.

Post-operatively, besides the care of either an open or closed abdomen, the patient will likely need nutritional support. Enteral (by mouth) feeding is the best, so placement of a feeding tube while in surgery should be considered. Certainly if the patient continues to vomit or enteral feeding is not appropriate, parenteral (IV) feedings should occur. Intravenous fluids, pain medication and antibiotics should be tailored to the patient's condition. The overall survival rate of septic peritonitis is about 25%, so owners should be given a guarded prognosis once the diagnosis is made.

DISEASES OF THE SMALL & LARGE INTESTINES

PROTEIN LOSING ENTEROPATHY

When the disease to the small intestine is severe, it can cause hypoproteinemia by allowing protein to leak into the gut lumen. Protein losing enteropathy (PLE) can occur for a myriad of reasons including: lymphangiectasia, parvovirus, intussusception, lymphosarcoma, lymphocytic-plasmacytic enteritis, inflammatory bowel disease or AHDS/HGE. The source of the protein loss is usually from the vasculature or the mucosal interstitial space. If left untreated, the final outcome of PLE is the loss of globulin and albumin which results in a decrease in intravascular oncotic pressure, development of abdominal and pleural effusion and peripheral edema. Generally PLE requires an intestinal biopsy to obtain a diagnosis. In cats, PLE is most commonly associated with GI lymphoma. Certain breeds such as the Yorkshire Terrier, Soft-Coated Wheaten Terrier, Basenji and Shar Pei are predisposed to PLE. Most animals with PLE will show a decrease in serum albumin, total protein and globulins. Once PLE is suspected, the cause must be diagnosed. Further bloodwork, fecal analysis, radiographs, ultrasound or biopsies may be indicated.

INFLAMMATORY BOWEL DISEASE

Inflammatory bowel disease (IBD) is a nondescript term which is used to characterize unknown causes of intestinal inflammation. The reality is there are many causes of IBD, and it is important to diagnose the cause in order to treat appropriately. Causes of chronic intestinal inflammation include: giardia, toxoplasmosis, lymphoma or histoplasmosis. There are three main forms of idiopathic IBD: lymphocytic plasmacytic enteritis (LPE), eosinophilic enteritis (EE) and granulomatous enteritis (GE). LPE is the most common form of idiopathic IBD. It is characterized by the increase of lymphocytes and plasma cells (inflammatory cells) that overpopulate the mucosa. Certain breeds are predisposed including the Basenji and German

Shepherd. LPE generally occurs in older animals. Although a definitive cause has not been defined, LPE is thought to be associated with an abnormal immune response to environmental stimuli. Signs range in severity, but generally include chronic vomiting, diarrhea and anorexia. Unfortunately, trying to differentiate between lymphoma and LPE can sometimes be very challenging. A diagnosis of LPE is dependent on an intestinal biopsy. Severe forms of LPE can lead to PLE, and this is particularly noted in the Basenji where a severe hereditary form has been identified. The second most common form of idiopathic IBD is EE. EE is similar to LPE, but instead, eosinophils infiltrate the mucosa. It is more commonly seen in younger animals. GE is the rarest form of idiopathic IBD and is characterized by the mucosa becoming infiltrated with macrophages forming granulomas.

Treatment for idiopathic IBD includes: dietary modification (usually hypoallergenic or bland diet), antibacterials, immunosuppressive drugs and anti-inflammatories. Though antibacterials are not necessarily “required” there is some evidence to support that the mucosa can become so severely damaged that small intestine bacterial overgrowth can occur. Metronidazole is also thought to have some immunosuppressive effects in addition to its antimicrobial activity. The most common immunosuppressive drugs include glucocorticoids (prednisolone, dexamethasone), cyclosporine and azathioprine. Immunosuppressive drugs are the number one treatment option for idiopathic IBD. Anti-inflammatories such as sulfasalazine or mesalazine can be used to help control intestinal inflammation. The absorption from the small intestines of these anti-inflammatory drugs may be nephrotoxic and should be used with caution.

PANCREATIC DISEASES

Pancreatitis (inflammation of the pancreas) is a disease that can be very frustrating because many times the cause is unknown. Pancreatitis can be divided into two different forms: chronic and acute. The most widely accepted theory as to why acute pancreatitis occurs is because autodigestion occurs (the pancreas literally starts to digest itself). This occurs when the pancreatic enzymes become activated within the small intestines through a series of events. Subsequently, this leads to the activation of the inflammatory process with the production of neutrophils, cytokines and free radicals.

Risk factors for developing pancreatitis are numerous including: nutritional factors (high fat diets), abdominal trauma, inflammatory bowel disease, hyperadrenocorticism, biliary duct obstruction, hypothyroidism and diabetes mellitus. Certain dog breeds seem more susceptible to chronic pancreatitis such as spaniels (Cockers and Cavaliers), collies and Boxers. Animals with pancreatitis can exhibit anorexia, vomiting and signs concurrent with severe abdominal pain such as walking with a hunched over appearance or hiding in cats. In severe cases the animal may present in hypovolemic shock.

The diagnosis of pancreatitis can be difficult. As veterinary medicine progresses more accurate serum/blood tests are becoming available. There are two main tests that can be submitted out to laboratories for a more conclusive diagnosis. Trypsin-like immunoreactivity (TLI) will detect serum trypsinogen which is only produced in the pancreas. However, it is reported that approximately only 30% of pancreatitis cases will have increased TLI values. The pancreatic lipase immunoreactivity (PLI) assay eliminates interference of other non-pancreatic origin lipase activities. This has proven to be the most accurate serum test for diagnosing pancreatitis currently available, but can take up to a week for the results. In-house bloodwork can be helpful as well, but rarely will yield a definitive diagnosis. Amylase and lipase levels were

formerly used by most veterinarians to diagnose pancreatitis. Unfortunately, many times these results were inaccurate. Amylase, although found in high concentrations in pancreatic cells, is not solely found there. Significant amylase is found in the small intestinal mucosal cells also. Lipase is not only found in the pancreas, but also in adipose tissue, gastric mucosal cells and duodenal mucosal cells. Both amylase and lipase can be elevated from GI disease or hepatic disease without the involvement of the pancreas. Interestingly, roughly 26% of dogs were found to be icteric when they had an acute pancreatitis. This occurs because bile (a yellow-green liquid secreted by hepatocytes made up primarily of salts, water and pigments) is secreted into the duodenum via the common bile duct, which is very close to the pancreatic duct. Inflammation in that area can cause secondary biliary blockage. Most recently, IDEXX introduced the canine SNAP® cPL™ Test, which measures canine pancreas-specific lipase. The test is easy and quick to use and boasts efficacy rates of greater than 90%. Certainly this is a good test to rule out pancreatitis quickly in dogs. If the test comes up positive, then pancreatitis should be highly suspected, and further diagnostics and/or treatment should be performed. Ultrasound is usually the diagnostic instrument of choice because it is non-invasive and quick. Unfortunately, false negatives can be common on ultrasound in cats.

The main treatment option for pancreatitis is intravenous fluid therapy. The fluids must be tailored to the patient's needs and may include isotonic crystalloids, colloids and plasma. Plasma should be given if DIC is suspected because it contains clotting factors and antithrombin III. Pain medication should be administered, because pancreatitis is known to be very painful. Historically, nutritional therapy was held for 5-7 days, however more research indicates it is better to start nutrition earlier. Enteral nutrition is the best option, but if the patient continues to vomit, parenteral nutrition may be needed. Antiemetic therapy should also be added to those patients that are vomiting.

References Available From The Author