ACUTE PANCREATITIS IN DOGS: AN UPDATE

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Acute vomiting – diagnostic approach

The first step in the approach to the acutely vomiting dog is to determine that vomiting and not regurgitation is present. Vomiting is associated with signs of nausea (depression, salivation, and frequent swallowing,) that is followed by abdominal contractions prior to the expulsion of material. Regurgitation is associated with esophageal disorders and occurs passively, usually associated with increased intrathoracic pressure that may be caused by excitement, activity, or changes in body position.

Once you have determined the dog is vomiting rather than regurgitating,, the next step is to determine if a self-limiting or life threatening problem is present. This assessment is crucial and must be based on a thorough history, careful physical examination, clinical experience and judgment, and a sound understanding of the differential diagnosis of acute vomiting. Dogs with acute pancreatitis can present with both types of vomiting. Animals should be considered to have a potential life-threatening problem if some of the following are present: Moderate or severe abdominal pain, lethargy, dehydration or pyrexia, enlarged distended bowel, frequent and severe diarrhea, hematemesis, frequent vomiting or increasing frequency of vomiting, signs of systemic disease, or puppies with an incomplete vaccination history. If a clear distinction cannot be reached, it is better to error on the cautious side and consider a potential life-threatening problem.

Dogs with a self-limiting problem require minimal diagnostic testing and symptomatic treatment, and often cease vomiting within 12-24 hours of initial presentation. A minimum data base for animals with self-limiting vomiting should include determination of packed cell volume and total solids, zinc sulfate fecal flotation, and digital rectal examination. Some common causes include acute gastritis or enteritis, dietary indiscretion, drug administration, toxin ingestion, foreign body ingestion, parasites, and coronavirus. Reclassification to life-threatening status may be indicated if an animal initially assessed as having self-limiting acute vomiting continues to vomit despite appropriate symptomatic therapy.

Life-threatening cases of acute vomiting require an in-depth diagnostic evaluation, vigorous symptomatic management, and often specific therapy directed at the underlying cause. The initial minimum data base for life-threatening acute vomiting includes a complete blood count, biochemical profile with amylase and lipase, urinalysis, zinc sulfate fecal flotation, and survey abdominal radiographs. After the initial evaluation, additional diagnostic studies may be indicated in some instances, such as upper GI endoscopy, upper GI barium series, abdominal

ultrasonography, ACTH response testing, or surgical exploration of the abdomen. Some common causes include acute gastritis, dietary indiscretion, hookworms, foreign body obstruction, intussusception, parvovirus, distemper, HGE, acute renal failure, acute liver failure, hypoadrenalcorticism, diabetes mellitus, and pyometra.

Acute pancreatitis

Acute pancreatitis commonly occurs in the middle-aged, obese female dog. Clinical signs include vomiting, diarrhea, abdominal pain, and fever. Historical association may be made with ingestion of a fatty meal or corticosteroid administration. Acute pancreatitis rapidly leads to severe dehydration (dry mucous membranes, loss of skin turgor, prolonged capillary refill time, or enophthalmos) and may progress to hypovolemic shock (tachycardia and weak peripheral pulses). In a recent necropsy study, 64% had pancreatic inflammation, many with chronic changes. Most of these dogs had another primary necropsy diagnosis, suggesting that chronic subclinical inflammation with lymphocytes may be an age related change. These findings question the utility of pancreatic biopsy as a gold standard for diagnosis.

The pathogenesis of AP is complex. It is a self-perpetuating auto-digestive process. As auto-digestion of the pancreas occurs, potent digestive enzymes are released into the parenchyma of the pancreas, blood vessels, and to the adjacent abdominal cavity. This causes severe hemodynamic alterations, localized inflammation, and can trigger disseminated intravascular coagulation. Depletion of circulating and tissue anti-proteases occurs. Vascular collapse develops due to a combination of the following: fluid loss from vomiting and diarrhea, release of vasoactive substances, release of cardiodepressant substances, or fluid sequestration within the abdominal cavity. Progression of the disorder may depend on preservation of pancreatic microcirculation, which can be maintained by fluid therapy.

Cases of AP can have inconsistent laboratory parameters. Diagnosis should not be based on any single test. Common changes include: leukocytosis with a left shift, elevated hematocrit, total protein, and prerenal azotemia (dehydration), elevated ALT and ALP, hypercholesterolemia, hyperglycemia, hypocalcemia, and lipemia. Classically, serum amylase, lipase, and trypsin-like immunoreactivity (TLI) should be elevated. However, elevations are not definitive for pancreatitis as amylase is contained in many tissues and lipase has recently been identified in the stomach. Amylase, lipase, and TLI depend on the kidney for elimination, thus prerenal azotemia due to dehydration from any cause of vomiting results in mild elevations. Some cases of AP have normal or only slightly elevated serum amylase, lipase, and TLI. In experimental AP, serum trypsin-like immunoreactivity (TLI) increases prior to amylase and lipase. Based on preliminary results, a new serum test is showing promise in diagnosing pancreatitis in dogs. The test, serum canine pancreatic lipase immunoreactivity (cPLI), was developed by Texas A&M researchers and immunologically measures

lipase from the pancreas. The test showed a sensitivity of 82% in the diagnosis of acute pancreatitis; these results are from a low number of cases, but are promising. A modification of this test using a monoclonal antibody and a recombinant antigen for calibration has been marketed by IDEXX as the Spec cPL. This test compares favorably with the cPLI and because of plate stability can be run daily with results rapidly reported. In a recent study of necropsied dogs with macroscopic evidence of pancreatitis the cPLI and SPEC cPL correlated and their overall sensitivity was 64%. IDEXX has also developed a in-house screening test (SNAP cPL) that has been shown to correlate with the Spec cPL. More data are needed, but these tests may be the most accurate serum tests for diagnosing acute pancreatitis in dogs.

Radiographic signs of AP are nonspecific and don't often contribute to diagnosis except by eliminating the presence of intestinal obstruction. Ultrasonographic evaluation of the abdomen can be very helpful and may identify a pancreatic mass or an enlarged hypoechoic pancreas that may surrounded by a hyperechoic rim. Pancreatic abscesses and cysts can also be identified.

Treatment
The therapeutic plan should prevent pancreatic secretion and manage hypovolemia while supporting pancreatic circulation. In severe cases, the dog should be maintained NPO and vigorous fluid therapy administered. Lactated ringers is an appropriate fluid to use at a volume necessary to correct dehydration, provide maintenance (44-66 ml/kg/day), and to replace losses due to vomiting and diarrhea. Potassium supplementation, 20 mEq/l KCl, is necessary to replace losses in diarrhea, vomitus, and urine and supplement the lack of food intake. Potassium supplementation should be based on measurement of serum potassium levels. Plasma transfusion (6-12 ml/kg) has been recommended to provide a fresh source of protease inhibitors. Mildly affected dogs may be held NPO and given fluids subcutaneously until the vomiting ceases for 12 hours. Vigorous pain control should be utilized, as pain may be one trigger for continued vomiting. Enteral nutrition should be administered as soon as reasonably possible. Use of a potent anti-emetic, such as maropitant, helps to reduce the frequency of vomiting quickly, allowing raster use of enteral feeding.

Treatment should continue until parameters used to make a diagnosis return to normal, often 3-5 days in moderately affected dogs. Gradual oral alimentation can be initiated. Initially, ice cubes or small amounts of water are frequently offered. If vomiting does not occur, small amounts of a bland diet can be frequently offered. This diet should be soft and low in fiber, highly digestible, high in carbohydrates, low in fat, and low in protein. Boiled rice, rice with chicken, low fat cottage cheese, or prescription diets such as i/d® (Hills Pet Products), EN® (Ralston Purina), or Low Residue (Iams) are effective. The size of the meals should be slowly increased and the frequency of feeding decreased

if vomiting does not recur. If the dog does not vomit for 3 days, the normal diet can be slowly added. Low fat diets have been recommended to prevent relapse.

If vomiting is severe, antiemetics can be used. Usually maropitant 1mg/kg q 24h is used. However, a phenothiazine, chlorpromazine 0.5 mg/kg q 4-6h, or metoclopramide, 0.2-0.4 mg/kg q 8h can be used. Because phenothiazines cause vasodilation they cannot be started until the dog has been rehydrated. Metoclopramide is contraindicated in cases with GI obstruction so obstruction should be eliminated prior to its use. If prolonged fluid therapy is necessary (7-10 days) total parenteral nutrition should be considered.

The prognosis for cases of AP is variable. Self-limiting cases respond to minimal therapy. Life-threatening cases warrant a guarded prognosis. Response to therapy in 3-5 days is a favorable prognostic sign. Dogs requiring intensive therapy for longer than 7 days carry a guarded prognosis. Because the etiology is unclear, recurrent bouts can occur.

Because the diagnosis of AP is difficult to prove, a thorough evaluation of other causes of acute vomiting, acute diarrhea, and abdominal pain should be performed. Classic findings of AP include: 1) acute vomiting, 2) cranial abdominal pain, 3) pyrexia, 4) leukocytosis with a left shift, 5) elevated serum amylase, lipase, cPLI, and SNAP cPLI and 6) ultrasonographic findings of an enlarged hypoechoic pancreas. Supportive findings include: 1) signalment 2) recent fatty meal, 3)corticosteroid administration, 4) lipemia, 5) hypocalcemia, 6) elevated ALT, ALP, and bilirubin, and 7) hypercholesterolemia.

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UPDATES IN GASTROENTEROLOGY
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INCREASED MUCOSAL PERMEABILITY, GASTITIS, EROSION, AND ULCER THERAPY

Increased mucosal permeability and erosion of the gastric mucosa commonly occur in dogs and cats with acute or chronic gastritis. Ulcers are uncommon in animals with gastritis but can be associated with nonsteroidal anti-inflammatory drug use. Back-diffusion of acid across damaged mucosa leads to further damage and retards mucosal healing. Stimulation of mast cells leads to elaboration of histamine, which caused further gastric secretion. Blood vessel damage reduces the mucosa's ability to heal. Further mucosal damage increases mucosal permeability further, allowing more gastric acid to back-diffuse, f\producing a self-perpetuating cycle. Reduction of gastric acid secretion, protection of ulcerated mucosa, or augmentation of cytoprotection promotes healing of erosions and ulcers.

H$_2$ Receptor Blockade

Drugs such as cimetidine (Tagamet), ranitidine (Zantac), famotidine (Pepsid), and nizatidine (Axid) block the H-2 receptor on the gastric parietal cell and decrease acid production. Cimetidine (5-10 mg/kg TID), ranitidine (2 mg/kg BID-TID), and famotidine, 0.5 mg/kg BID have been used most commonly in veterinary medicine. They can be given orally or parenterally and have not been commonly associated with adverse effects. Cimetidine can inhibit hepatic cytochrome P-450 enzymes, potentially interfering with the metabolism of other drugs. Nizatidine, 5 mg/kg SID (this dosage has not been well established), has not been used as frequently in veterinary medicine, but is also effective. All four of these drugs are now available over the counter in smaller dosage forms than prescription strength, making treatment of cats and small dogs easier. Elixirs are available for cimetidine, ranitidine, and famotidine. The drugs have a rapid onset of action. However, recent studies have shown that H$_2$ blockers are less effective in reducing gastric acid secretion in normal dogs and cats than in humans. These studies have shown that gastric pH remains below 3 for more than 12 hours/day, well below the guidelines utilized in humans to heal gastric lesions or treat gastroesophageal reflux. On the other hand, PPI's have performed much better, questioning the effectiveness of H$_2$ blockers in dogs and cats.

Proton Pump Inhibitors (PPI's)

PPI's inhibit the action of the proton pump at the apical portion of the parietal cell that exchanges H+ for luminal K+, thus preventing secretion of acid. As a weak base PPI's accumulate in the acid compartment of the parietal cell, necessitating only SID administration in humans. Omeprazole (Prilosec) is the most commonly used PPI in veterinary medicine. The recommended dose is 1.0 mg/kg SID. The enteric-coated granules (20 mg) are packaged in gelatin capsules to resist degradation by gastric acid. If less than one capsule is to be administered (20 mg), the granules should be repackaged in gelatin capsules. Zegerid is an omeprazole powder that is mixed with bicarbonate to protect the drug from gastric acid. It can be divided into smaller doses. Another PPI, lansoprazole (Prevacid) granules can be mixed in an acid juice, such as apple juice and administered. Other PPI's such as pantoprazole (Protonix), rabeprazole (Aciphex), esomeprazole (Nexium) must be reformulated into a form that protects the drug from gastric acid damage, mixed with 8.4 % bicarbonate or compounded with cod liver oil. Omeprazole also inhibits hepatic p-450 enzymes so interference with the metabolism of other drugs is possible. However, these drugs have not been associated with frequent adverse reactions in dogs and cats. Omeprazole, pantoprazole, and lansoprazole are available over the counter at lower dosages than in the prescription dugs.

Several recent studies in normal dogs and cats have shown that PPI's in dogs are superior at inhibiting acid secretion than H$_2$ blockers. or cats. Additional studies have shown that BID administration further improves acid suppression. Diarrhea may develop with BID administration, so some animals may not be able to be treated with BID dosages. My concern

about these recent studies is that they have utilized acid suppression guidelines from humans which have been developed for the treatment of gastric and duodenal ulcers and gastroesophageal reflux. Most animals treated with acid suppressors don't have ulcers, they have increased gastric mucosal permeability or gastritis. It is unknown what degree of acid suppression is necessary to heal these disorders in dogs and cats. Additionally, there is a paucity of studies in dogs and cats with clinical disease, making interpretation of physiologic studies in normal animals difficult.

Sucralfate

Sucralfate (Carafate) is a sulfated disaccharide that forms an adherent gel and binds to an ulcer crater, protecting it from acid and pepsin. It also stimulates the synthesis of prostaglandin, increases mucosal cytoprotection, and binds epithelial growth factor at the ulcer, where it stimulates cellular proliferation. It has been shown to be as effective as H-2 receptor blockers in healing ulcers in humans. Because sucralfate can bind other drugs, medications should be given 1-2 hours prior to sucralfate administration. The recommended dose is 1 gm/25 kg TID-QID in dogs and 0.25 gm TID in cats. Because absorption is minimal, toxicity is uncommon. Long-term use may lead to constipation because of its aluminum content. There is no evidence to support that combination therapy with an H-2 receptor antagonist provides added benefit compared to therapy with either sucralfate or an H-2 blocker alone.

Sucralfate is also effective to treat esophagitis because of its ability to coat ulcerated mucosa. The suspension form is necessary for this indication.

Misoprostol

Misoprostol (Cytotec) is a synthetic prostaglandin that prevents or heals ulcers associated with NSAID administration by directly increasing mucosal cytoprotection. The suggested dose is 3µg/kg TID. The most common side effect is diarrhea although it can also cause abortion. Its major indication is preventing GI mucosal injury in dogs with arthritis that require long-term NSAID therapy. It can also be used to treat cases of GDUD caused by NSAIDS.

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PROBIOTICS

Probiotics are live bacteria that confer a health benefit to the host. Common bacteria include lactobacilli, bifidobacteria, and enterococci. In humans a daily dose is often 5-10 million. To be effective viability must be maintained throughout production, storage, distribution, passage through the upper GI tract into the colon. Many commercially available products do not survive transit into the colon and are not as effective as "advertised". The bacteria should be able to be cultured from the feces during treatment, but will usually disappear once oral administration ends. The bacteria must be nonpathogenic and not transmit antibiotic resistance.

Probiotic bacteria have been reported to have many beneficial effects on the host including conditioning the immune system, synthesizing B vitamins, producing digestive enzymes, producing antibacterial factors, competing with pathogens for adhesion sites and nutrients, enhancing epithelial repair, increasing mucus production, decreasing luminal pH, and protecting tight junctions. However, all probiotics do not do all of the above.   In humans some probiotics have been shown to be beneficial in acute infectious diarrhea, prevention of antibiotic associated diarrhea, pouchitis, cow's milk allergy, IBD, and irritable bowel syndrome. Many probiotics that have been independently tested did not contain the the type and numbers of live bacteria listed on the label. Caution should be utilized when selecting a probiotic, utilizing those with excellent quality control and published research demonstrating numbers and types of live bacteria within the product. Currently there is accumulating evidence demonstrating benefits of probiotics in dogs and cats with diarrhea, including dogs with IBD.

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THE DIAGNOSTIC UTILITY OF ABDOMINAL ULTRASOUND IN DOGS WITH CHRONIC VOMITING AND WITH CHRONIC DIARRHEA

Abdominal ultrasonography has recently been added to the diagnostic plan for many dogs and cats with chronic vomiting or chronic diarrhea. Ultrasound has been shown to be very helpful in animals with a mass lesion, especially neoplasia. An ultrasound guided fine needle aspirate or tru-cut biopsy can be performed. Ultrasound has also been shown to helpful in cases with chronic pancreatitis. Other advantages of performing ultrasound include: being noninvasive, imaging of the liver and biliary system, imaging of the small and large bowel and mesenteric lymph nodes, and assessment of the layers of the GI tract and its motility. Disadvantages include the need for expensive equipment and specialized training, interference by gas within the GI tract, and difficulty in imaging the pancreas.

Two studies have been performed in which the diagnostic utility of abdominal ultrasound in dogs with chronic vomiting or chronic diarrhea has been evaluated. A single radiologist performed each abdominal ultrasound. Two internists, who did not directly participate in case management, reviewed each medical record. In each case, the contribution the ultrasound made towards the final diagnosis was assessed and scored from 1-5, based on the following scale:

1. Diagnosis was obtained via ultrasonography (including ultrasound-guided aspirate or biopsy). Additional biopsy via endoscopy or exploratory celiotomy was not necessary.
2.   Ultrasonography provided data that suggested endoscopy was not indicated and exploratory celiotomy should be performed to obtain a diagnosis.

Ultrasonography suggested how to obtain a tissue biopsy, making it very important for diagnosis.
3. Ultrasonography provided important diagnostic information that helped assess other data, including endoscopic findings. Ultrasonography was important in arriving at a diagnosis.
4. Ultrasonography provided descriptive information that did not affect assessment of other data obtained via endoscopy or exploratory celiotomy. The same diagnosis would have been reached without performing ultrasonography.
5. Ultrasonography provided conflicting information that did not support, or may have hindered obtaining the final diagnosis.

In the group of dogs with chronic vomiting, the following factors were associated with a higher diagnostic utility of abdominal ultrasound: presence of weight loss, higher percentage of body weight lost, increasing age, increasing duration of vomiting, an increased frequency of vomiting/week, and a final diagnosis of GI lymphoma or gastric adenocarcinoma. Based on diagnostic utility scores, abdominal ultrasonography was vital or beneficial to obtaining a diagnosis in 22.5% of cases, not helpful in 68.5%, and of marginal value in 9%. Other benefits of ultrasound, unrelated to vomiting, were identified in 12% of dogs. Considering all contributions to case management, abdominal ultrasound was considered helpful in 27% of dogs with chronic vomiting.

In the group of dogs with chronic diarrhea the following factors were associated with a higher diagnostic utility of abdominal ultrasound: the presence of weight loss, palpation of an abdominal or rectal mass on initial physical examination, localization of diarrhea to mixed bowel (vs. large bowel), diseases that commonly have mass lesions that should be visible on ultrasound examination, and a clinical diagnosis of GI neoplasia. Based on diagnostic utility scores, abdominal ultrasonography was vital or beneficial to obtaining a diagnosis in 15% of cases, not helpful in 68%, and of marginal value in 17%. Other benefits of ultrasound, unrelated to diarrhea, were identified in 17% of dogs. Considering all contributions to case management, abdominal ultrasound was considered helpful in 25% of dogs with chronic diarrhea.

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MEDICATION-INDUCED ESOPHAGEAL STRICTURES IN CATS
Doxycycline is commonly used in cats to treat many potentially infectious diseases. The drug is acidic and can be caustic to esophageal epithelial cells. It may accumulate within epithelial cells, where it can decrease protein synthesis and potentially decrease mucosal repair. Esophagitis can progress to stricture formation after doxycycline administration in cats. These strictures result in dramatic reduction of the esophageal lumen and severe regurgitation and dysphagia. Clinical signs usually develop within 7-10 days of administration of doxycycline. Treatment requires repeated endoscopic balloon dilation, with is an expensive and invasive procedure. Doxycycline induced esophageal strictures in cats usually occur in the proximal esophagus. Stricture diameter is often very small (often 1-5 mm) at initial diagnosis, smaller than strictures associated with anesthesia and gastroesophageal reflux. Usually re-stricturing is a major problem and affected cats often require more frequent dilations than cats with strictures due to other causes. Intralesional corticosteroid injection may reduce the frequency of repeated dilations. Post-dilation treatments often include and H2 blocker, metoclopramide, sucralfate, prednisone, and in some cats placement of a PEG tube. Oral feeding with a liquid or blenderized diet is often necessary after dilation.

Two recent studies in normal cats have clearly demonstrated that transport of capsules and tablets through the esophagus after "dry" swallows was very delayed. This delay is thought to be responsible for the development of esophagitis and subsequent esophageal stricture formation. As many sick cats are anorectic and potentially dehydrated, it is possible that esophageal transport of tablets and capsules may actually be slower than demonstrated experimentally. To aid transport of tablets and capsules and avoid stricture formation, a 6 ml water flush or a small amount of food should always follow doxycycline administration in cats. Doxycycline should be discontinued at the first signs of regurgitation or dysphagia. Recently esophagitis and strictures have also been seen with clindamycin.

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