Vomiting

Melanie Werner
Dr. med. vet.,
DECVIM-CA
(Internal Medicine)

Pathophysiology

Vomiting, or emesis, is a common clinical presentation in veterinary practice and can be a distressing experience for both dogs and their owners. It is an intricate physiological process involving a combination of neurological, gastrointestinal, and metabolic pathways. Understanding the pathophysiology and clinical signs of vomiting is essential for a small animal veterinarian to perform a precise diagnosis and plan effective management.

The pathophysiology of vomiting is a complex reflex action initiated in the emetic center of the brain, comprising several nuclei situated in the medulla oblongata. These nuclei house various receptors, including serotonergic (5HT1), adrenergic (alpha-2), and neurokinergic (NK1). Activation of these receptors triggers the complex reflex action of vomiting, serving as a protective mechanism against the ingestion of potentially harmful substances. The emetic center receives input from various sources, including indirect activation through humoral pathways via the chemoreceptor trigger zone (CRTZ) or direct stimulation via neural pathways originating from the gastrointestinal tract, cerebral cortex, vestibular system, or other parts of the body. The CRTZ, located in the postrema area on the floor of the fourth ventricle, lacks a blood-brain barrier, making it sensitive to detecting chemical stimuli in the bloodstream. Both endogenous (e.g., uremic or hepatoencephalic toxins resulting from renal failure or hepatic encephalopathy) and exogenous substances (e.g., drugs, environmental toxins, or endotoxins) can reach the CRTZ. The detection of these substances by the CRTZ signals the need for emesis as a protective response.

The vomiting reflex can also be triggered by neural stimulation of the emetic center through various pathways, including afferent vagal, sympathetic, vestibular, and cerebrocortical paths. Disorders affecting the gastrointestinal tract can stimulate vomiting by triggering the release of serotonin from enterochromaffin cells located in the gut mucosa. The released serotonin binds to 5HT3 receptors on afferent vagus nerves, leading to the stimulation of emesis.[1] Therefore, conditions causing gastrointestinal (GI) inflammation or obstruction may result in vomiting due to an increase in serotonin release. Furthermore, the coordination of various muscles during vomiting involves the pharynx, diaphragm, and abdominal muscles. The diaphragm contracts to increase intra-abdominal pressure, while the abdominal muscles contract to expel the gastric or duodenal contents. Simultaneously, the lower esophageal sphincter relaxes to allow the contents to move upward, and the larynx closes to prevent aspiration of vomitus, showing the importance of the reflex.[2]

In addition to the central and peripheral mechanisms described, vomiting may also occur due to increased intracranial pressure or following stimulation of the vestibular apparatus, commonly seen in conditions like vestibular syndrome.

Clinical signs

Vomiting refers to the forceful expulsion of gastric or duodenal contents through the oral cavity. The nature of vomitus can offer clues to the pathophysiology. For instance, the presence of undigested food observed several hours or even a day after ingestion suggests delayed gastric emptying due to gastric outflow obstruction or hypomotility. Conversely, bile-laden vomit indicates a patent gastric outflow tract with an open pylorus, signifying intact small intestinal motility. Blood-tinged vomit, whether fresh blood or coffee-ground appearance, raises suspicion of small gastrointestinal or esophageal erosions, ulcers, or potential bleeding neoplasia.[3] A thorough physical examination during a vomiting episode may reveal abdominal pain and distension. Abdominal pain can be indicative of inflammatory processes such as pancreatitis or obstructive lesions. Frequent or prolonged vomiting can lead to dehydration and electrolyte imbalances, manifested by dry mucous membranes, sunken eyes, and reduced skin turgor. Consequent weakness and lethargy may further complicate the clinical presentation. Chronic vomiting may result in weight loss, as the dog experiences nutrient and caloric deficits over time. If an intracranial process is the cause of vomiting, neurological signs may accompany vomiting, such as circling, head pressing, or

seizures. The presence of fever is rare in the vomiting dog but may signal systemic infections or inflammatory conditions such as pancreatitis or hepatitis, warranting vigilant monitoring and further workup. Moreover, it is critical to distinguish between regurgitation and emesis. Regurgitation is characterized by effortless expulsion of undigested food from the esophagus, which points towards esophageal dysfunction or obstruction, while emesis involves active retching and abdominal contractions, indicating underlying gastric or intestinal disturbances. For aspects helping to differentiate vomiting and regurgitation, see Table 1.1.

TABLE 1.1 Differentiation of regurgitation and vomiting.

Aspect	Regurgitation	Vomiting
Definition	Effortless expulsion of undigested food from the esophagus, passive	Forceful expulsion of gastric or duodenal contents, coordinated reflex
Clinical significance	Points towards esophageal issues	Suggests underlying gastric, intestinal, metabolic, visceral, or neurological causes for the clinical sign
Associated actions	No retching or abdominal contractions	Active retching and abdominal contractions
Nausea and salivation	No	Common
Presence of bile	No	Possible
Presence of fresh blood	Possible	Possible
Presence of coffee-ground-like digested blood	No	Possible
Amount of material	Variable	Variable
Timing after eating	Variable	Variable
Risk of aspiration	High	Low

Diagnostic approach

Establishing whether the dog is vomiting or regurgitating is crucial. This is a critical first step, as the causes and subsequent diagnostic pathways for vomiting and regurgitation are significantly different. Remember, vomiting is a reflex, whereas regurgitation is passive, so with vomiting, there is usually evidence of nausea, and abdominal contractions precede the expulsion of gastric or duodenal contents. Regurgitation typically occurs without warning and involves the expulsion of food or fluid from the esophagus without abdominal effort.[4] Owners sometimes confuse coughing with vomiting, but the history should make this clear.

The diagnostic workup of a dog presenting with vomiting requires a meticulous and systematic strategy and is summarized in **Algorithm 1.1**.

Algorithm 1.1 Note 1 Begin with a thorough history taking and comprehensive physical examination. The veterinary practitioner should pay attention to the character of the vomitus, frequency and duration of vomiting, and any relationships to eating or drinking. Differentiating chronic and acute vomiting (persisting for over 7 days) is crucial, as it can provide valuable insights into the underlying etiology. Other relevant history might include previous episodes of vomiting, concurrent diarrhea, changes in appetite or drinking habits, weight loss, or behavioral changes.

Algorithm 1.1 Note 2 The dietary history is equally important, encompassing any recent changes, brand and type of food used, or the opening of a new food bag or can. The history should also include any potential exposure to toxins, foreign body ingestion, access to garbage, or feeding of table scraps.

Algorithm 1.1 Note 3 The physical examination includes general observations about the dog's body condition and hydration status, followed by a systematic examination. Abnormalities such as abdominal pain or the presence of a mass could provide clues to the underlying cause of vomiting.

Algorithm 1.1 Note 4 Laboratory tests are invaluable in the initial evaluation, including a complete blood count (CBC), serum biochemistry profile, specific pancreatic lipase (cPLi or DGGR-lipase) and urinalysis. These tests provide a general health assessment and may identify underlying systemic or metabolic diseases, such as kidney disease or liver function abnormalities, diabetic ketoacidosis, electrolyte imbalances or pancreatitis, which might be causing the vomiting. Moreover, especially if additional diarrhea is present, fecal examination for parasites or specific tests for infectious diseases (e.g., parvovirosis or leptospirosis) should be undertaken.[9,10]

Algorithm 1.1 Note 5 Blood gas analysis, although not essential in all cases, can help to identify metabolic hypochloremic alkalosis as an indicator for a potential gastric outflow obstruction.

Algorithm 1.1 Note 6 Imaging studies, including radiographs and abdomen ultrasound, are often the next step in the diagnostic process. Specific markers can be utilized in these imaging modalities when pyloric or intestinal obstruction is suspected. These findings may prompt further investigation with contrast studies or ultrasound. On ultrasound examination, the pylorus can be assessed for dilation, wall thickness, and the presence of intraluminal contents. Intestinal obstruction can also be visualized through ultrasound, with the identification of distended loops of the intestine, fluid-filled or gas-filled segments. Changes may also be seen in the liver, pancreas or other abdominal organs.[6,7]

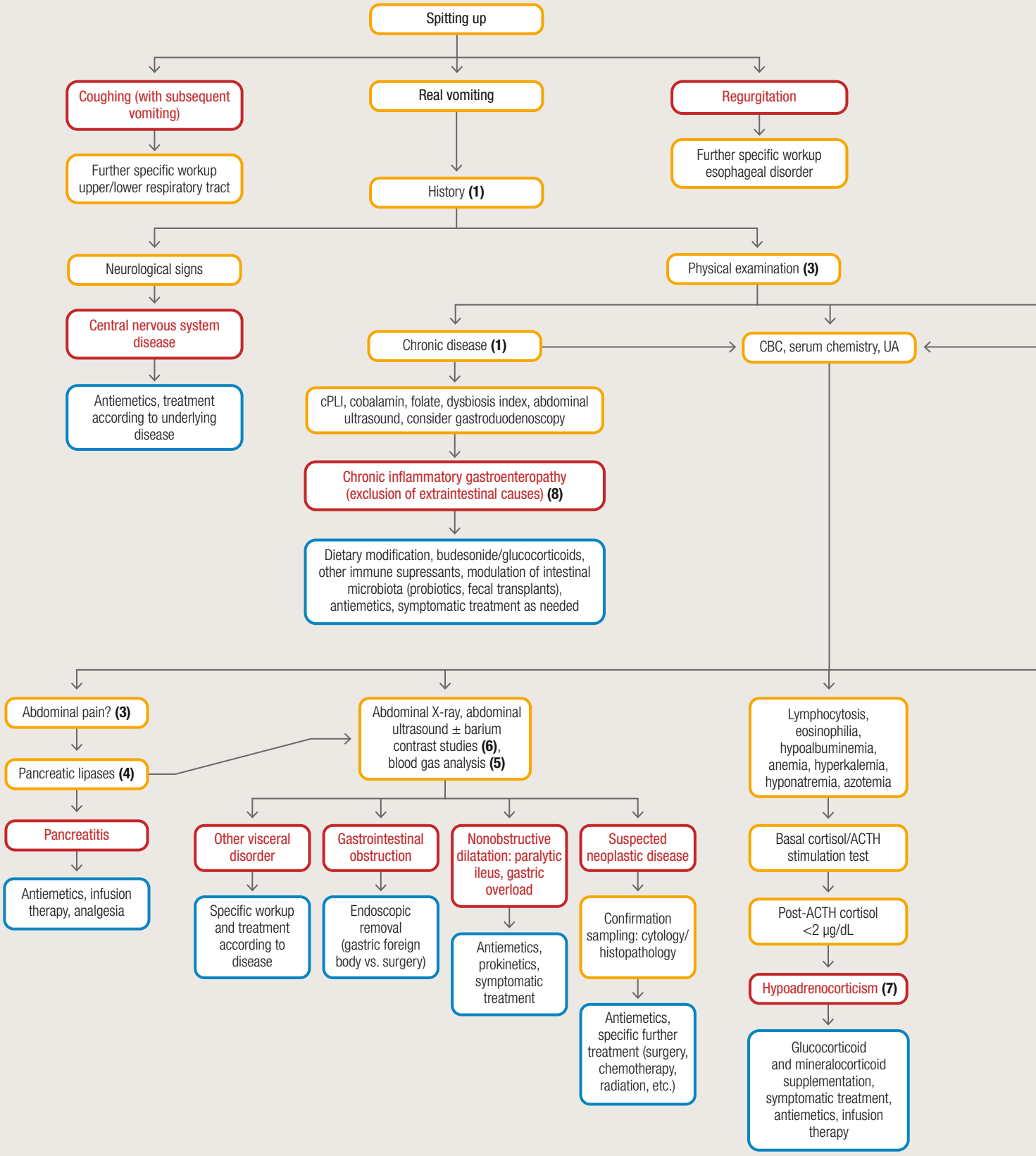
Algorithm 1.1 Note 7 Depending on the initial findings, more specific diagnostic tests might be required. These could include workup in the direction of other uncommon diseases causing vomiting (e.g., hypoadrenocorticism).[8]

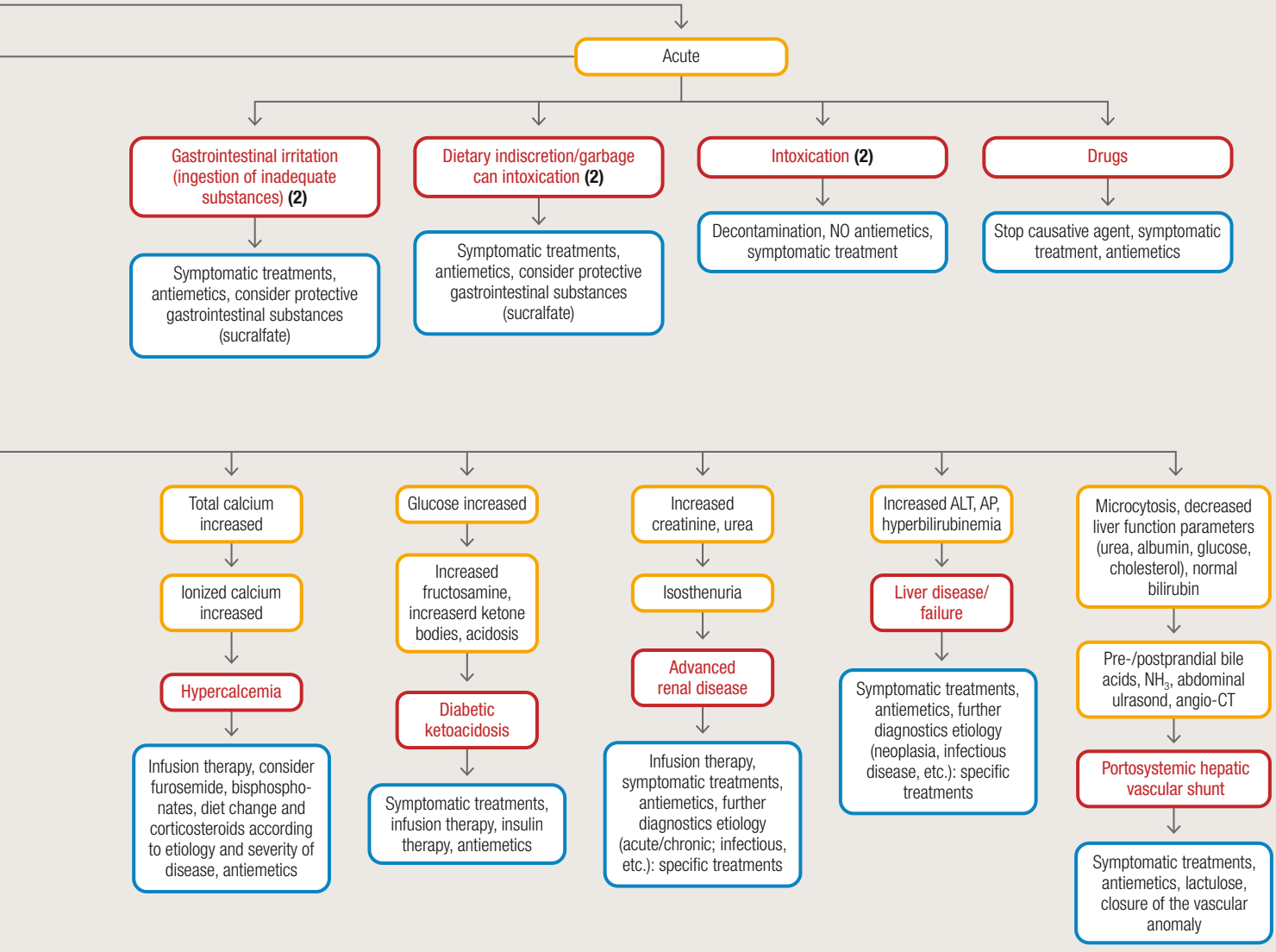
Algorithm 1.1 Note 8 Endoscopy may be indicated in chronic cases when extraintestinal diseases have been ruled out to visualize the GI tract directly (more in the chronic cases) and biopsy to diagnose conditions like chronic gastritis, chronic enteropathy, or neoplasia. Measuring cobalamin and folate and evaluating the dysbiosis index can give hints on any intestinal involvement in the disease in chronic cases.[11–13]

In conclusion, understanding the pathophysiology, recognizing the clinical signs, and adopting a systematic diagnostic approach is fundamental for effectively managing dogs with vomiting. Although vomiting is a common clinical sign, it can often be a challenge to determine its cause due to the wide variety of underlying conditions. Therefore, a meticulous, organized, and patient-centered approach is critical to reaching an accurate diagnosis and formulating an effective treatment plan, ultimately improving the quality of life for the affected canine patients.

ALGORITHM 1.1

Diagnostic workup of the vomiting dog.




Acute
Gastrointestinal irritation (ingestion of inadequate substances) (2)
Symptomatic treatments, antiemetics, consider protective gastrointestinal substances (sucralfate)
Dietary indiscretion/garbage can intoxication (2)
Symptomatic treatments, antiemetics, consider protective gastrointestinal substances (sucralfate)
Intoxication (2)
Decontamination, NO antiemetics, symptomatic treatment
Drugs
Stop causative agent, symptomatic treatment, antiemetics
Total calcium increased
Ionized calcium increased
Hypercalcemia
Infusion therapy, consider furosemide, bisphospho-nates, diet change and corticosteroids according to etiology and severity of disease, antiemetics
Glucose increased
Increased fructosamine, increaserd ketone bodies, acidosis
Diabetic ketoacidosis
Symptomatic treatments, infusion therapy, insulin therapy, antiemetics
Increased creatinine, urea
Isosthenuria
Advanced renal disease
Infusion therapy, symptomatic treatments, antiemetics, further diagnostics etiology (acute/chronic; infectious, etc.): specific treatments
Increased ALT, AP, hyperbilirubinemia
Liver disease/ failure
Symptomatic treatments, antiemetics, further diagnostics etiology (neoplasia, infectious disease, etc.): specific treatments
Microcytosis, decreased liver function parameters (urea, albumin, glucose, cholesterol), normal bilirubin
Pre-/postprandial bile acids, NH_3, abdominal ulrasond, angio-CT
Portosystemic hepatic vascular shunt
Symptomatic treatments, antiemetics, lactulose, closure of the vascular anomaly
Diagnostic protocol
Diagnosis
Treatment

References

1. Fukui H, Yamamoto M, Sato S. Vagal afferent fibers and peripheral 5-HT3 receptors mediate cisplatin-induced emesis in dogs. *Jpn J Pharmacol* 59(2):221-226, 1992.
2. Washabau RJ. Vomiting. In Washabau RJ, Day MJ (editors). Canine and Feline Gastroenterology [E-book]. Saint Louis, W.B. Saunders, 2013, pp. 167-173.
3. Ettinger SJ, Feldman EC, Côté E. Textbook of Veterinary Internal Medicine-Inkling E-Book. Elsevier Health Sciences, 2016.
4. Kook PH. Esophagitis in cats and dogs. *Vet Clin North Am Small Anim Pract* 51(1):1-15, 2021.
5. Boag AK, Coe RJ, Martinez TA, Hughes D. Acid-base and electrolyte abnormalities in dogs with gastrointestinal foreign bodies. *J Vet Intern Med* 19(6):816-821, 2005.
6. Hayes G. Gastrointestinal foreign bodies in dogs and cats: A retrospective study of 208 cases. *J Small Anim Pract* 50(11):576-583, 2009.
7. Aronson LR, Brockman DJ, Brown DC. Gastrointestinal emergencies. *Vet Clin North Am Small Anim Pract* 30(3):555-579, 2000.
8. Hauck C, Schmitz SS, Burgener IA, Wehner A, Neiger R, Kohn B, et al. Prevalence and characterization of hypoadrenocorticism in dogs with signs of chronic gastrointestinal disease: A multicenter study. *J Vet Intern Med* 34(4):1399-1405, 2020.
9. Mylonakis ME, Kalli I, Rallis TS. Canine parvoviral enteritis: an update on the clinical diagnosis, treatment, and prevention. *Vet Med Auckl NZ* 7:91-100, 2016.
10. Mazzaferro EM. Update on canine parvoviral enteritis. *Vet Clin North Am Small Anim Pract* 50(6):1307-1325, 2020.
11. Kather S, Grützner N, Kook PH, Dengler F, Heilmann RM. Review of cobalamin status and disorders of cobalamin metabolism in dogs. *J Vet Intern Med* 34(1):13-28, 2020.
12. Heilmann RM, Steiner JM. Clinical utility of currently available biomarkers in inflammatory enteropathies of dogs. *J Vet Intern Med* 32(5):1495-1508, 2018.
13. AlShawaqfeh MK, Wajid B, Minamoto Y, Markel M, Lidbury JA, Steiner JM, et al. A dysbiosis index to assess microbial changes in fecal samples of dogs with chronic inflammatory enteropathy. *FEMS Microbiol Ecol* 93(11), 2017.