Received: 10 November 2021 | Revised: 2 March 2022 | Accepted: 8 March 2022

DOI: 10.1111/vru.13123

ORIGINAL INVESTIGATION

WILEY

CT features of confirmed and presumed gastric wall edema in dogs

Masahiro Murakami[1] | **Hock Gan Heng**[1] | **Mario Sola**[2]

[1]Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Purdue University, West Lafayette, Indiana, USA

[2]Department of Comparative Pathobiology, College of Veterinary Medicine, Purdue University, West Lafayette, Indiana, USA

Correspondence
Masahiro Murakami, Department of Veterinary Clinical Sciences, College of Veterinary Medicine, 625 Harrison Street, Purdue University, West Lafayette, IN 47907, USA.
Email: mmuraka@purdue.edu

Abstract

Postcontrast computed tomographic (CT) characteristics of gastrointestinal (GI) wall edema in humans have been described as GI wall thickening with a thickened submucosal layer and thin enhanced inner and outer layers. Published studies describing CT features of gastric wall edema in dogs are currently lacking. The aim of this retrospective, case series was to describe CT features of gastric wall edema in a group of dogs. Medical records were searched for dogs with postcontrast abdominal CT scans and a diagnosis of gastric wall edema based on histopathology (group I) or CT characteristics consistent with those reported in humans (group II). Clinical diagnosis, mean serum albumin concentration, and histopathological diagnosis were recorded. The following CT characteristics were recorded: numbers of wall layers, attenuation and contrast enhancement, presence of blood vessels, locations, distribution, and thickness. Twelve dogs (3 in group I and 9 in group II) were included. The most common clinical finding was hypoalbuminemia. In group I, a well-defined three-layer appearance with a non-enhancing fluid-attenuating middle layer was observed in three dogs and thin blood vessels in the middle layer in two dogs. In group II, nine dogs had a three-layer appearance with a non-enhancing fluid-attenuating middle layer. Locations of gastric wall thickening were diffuse in two, focal concentric in six, and focal asymmetric in four dogs. Findings supported including gastric wall edema as a differential diagnosis for dogs with hypoalbuminemia and CT characteristics of a three-layer appearance in the gastric wall, with a non-enhancing fluid-attenuating middle layer and thin blood vessels.

KEYWORDS
canine, gastric wall thickening, hypoalbuminemia, submucosal edema

Abbreviation: ACVR, American College of Veterinary Radiology; GI, gastrointestinal.

1 | INTRODUCTION

Computed tomography (CT) of the gastrointestinal (GI) tract is becoming common in dogs,[1,2] and is an established tool for the detection and characterization of GI abnormalities in humans.[3,4] However, distinct GI wall layering is not easily identifiable on CT due to reduced resolution compared to ultrasonography.[5–7] In post-contrast CT, the highly vascularized mucosal layer is the most strongly enhancing layer of the GI wall and may appear as a distinct layer in humans and dogs.[3,8] The less vascularized submucosal and muscularis layers are rarely distinguished on post-contrast CT unless they are edematous, hemorrhagic, or infiltrated by fat.[3,9] In cases with GI wall edema in humans, two or three concentric and alternating attenuation layers have been observed.[9,10] The three-layer appearance was described as a thin enhancing inner and outer layer and a thick poorly enhancing middle layer, and a two-layer appearance without a strong enhancement of the inner layer.[9,10]

The authors have occasionally observed three-layer gastric wall thickening in postcontrast CT with a thin enhancing inner layer, thick hypoattenuating middle layer, and an outer hyperattenuating layer in dogs, similar to the reported gastric wall edema in humans. In veterinary medicine, precise CT features or diagnostic criteria of gastric wall edema are lacking,[11,12] and we have therefore been using human CT features to speculate gastric wall edema in dogs.

The goals of the current study were to (i) describe the CT features of gastric wall edema with histological confirmation in dogs, (ii) subjectively compare CT features of dogs with histologically confirmed gastric wall edema and dogs with CT diagnosis of presumed gastric wall edema without histological confirmation, and (iii) describe relationships between clinical diagnoses and the locations of gastric wall edema.

2 | MATERIALS AND METHODS

2.1 | Experimental design and case selection criteria

This was a retrospective case series study design. The Purdue University Veterinary Teaching Hospital (PUVTH) Medical Record database from January 1, 2017, to June 1, 2020, was searched to identify dogs that had abdominal CT including the entire stomach, and had histological confirmation of gastric wall edema (Group I), and had gastric wall edema listed as one of the differential diagnoses in CT reports (Group II). Final decisions for subject inclusion or exclusion were made by an American College of Veterinary Radiology (ACVR) board-certified veterinary radiologist (M.M.). Clinical diagnosis and mean serum albumin concentrations (if performed within 24 h of the CT study) were recorded by the same radiologist. Due to the retrospective study design, no institutional animal care approval was required. However, all owners signed a consent form for use of images and medical information.

2.2 | CT image analyses

Pre- and postcontrast CT of the gastric wall and the rest of the abdomen in the form of digital files of groups I and II were reviewed by one ACVR-certified veterinary radiologist (M.M.) and one ACVR and European College of Veterinary Diagnostic Imaging (ECVDI)-certified veterinary radiologist (H.G.H.) by consensus using an image viewing workstation (Osirix MD: Pixmeo, Geneva, Switzerland). Dogs with gastric wall neoplasia (gastric wall thickening with loss of wall layering and histological confirmation of neoplasia) or suboptimal CT image quality of the gastric wall due to motion artifact (which hampers evaluation of wall layering) were excluded from the present study. Additionally, in group II, dogs were excluded if CT findings of the gastric wall were different from the CT features of gastric wall edema in group I. This exclusion was made after the review process of all cases and the readers were not aware of Group I results when Group II review was performed. Computed tomography criteria of the gastric wall lesions were evaluated: (i) numbers of distinct wall layers in pre- and postcontrast CT studies (single-layer, two-layer, or three-layer), (ii) attenuations (Hounsfield Unit: HU), and contrast enhancement of thickened gastric wall (yes or no), (iii) presence or absence of small blood vessels in the gastric wall, (iv) distribution (diffuse, focal concentric, focal asymmetric, or multifocal) and locations (fundus, body, and pyloric part) of the thickened gastric wall, (v) distribution of lesions, (vi) thickness (mm) of the gastric wall at the site of gastric wall edema, (vii) abnormality adjacent to the focal thickening if present. The HU of each layer in the pre- or postcontrast study was recorded if multiple layers were recognized, however, HUs were not evaluated in thin layers that were too thin to accurately measure HU due to partial volume averaging artifact. Circular regions of interest (ROIs) were used to measure the HU of the thick middle/outer layer. Sizes of the ROIs varied among individual patients since the largest ROIs in the thick middle/outer layer were placed without affecting adjacent layers (Figure 1). For each dog, HU was measured three times, and the average HUs were calculated. Each ROI placement was made by consensus of the ACVR-certified veterinary radiologist (M.M.) and the ACVR and ECVDI-certified veterinary radiologist (H.G.H.). Any enhancing vasculature visible on the postcontrast study was avoided when measuring the HU. An increase of more than 20 HU or visibly recognizable enhancement between pre- and postcontrast CT study was considered as the presence of contrast enhancement.[13–15]

Regions of the stomach wall evaluated were defined as follows: gastric fundus was dorsal to the level of the cardia: pyloric part was aborad to the level of the angular incisure: and gastric body was between the gastric fundus and the pyloric part.[16] Gastric wall thickening was considered as diffuse if the entire gastric wall was thickened. Single continuous thickening of the gastric wall involving multiple parts of the stomach (for example, continuous thickening through the gastric body and the pyloric part) was considered as a focal thickening, and multiple separated thickenings were multifocal. Focal concentric was used if the gastric wall of that location, such as fundus, body, or pyloric part, was circumferentially thickened. The focal asymmetric gastric wall

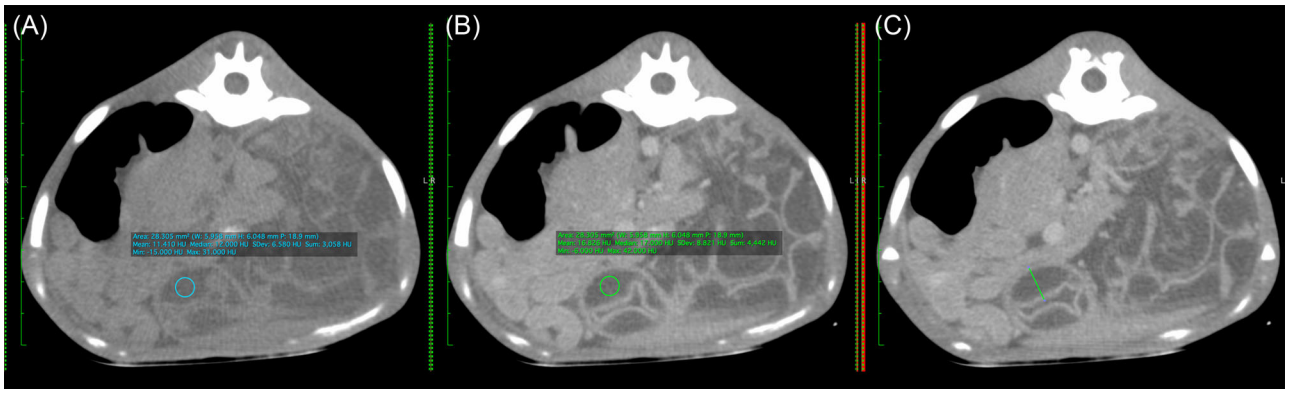

FIGURE 1 Examples for placement of ROIs in pre- (A) and postcontrast (B,C) transverse CT images in a dog with gastric wall edema in soft tissue window (window level: 40 HU, window width: 350 HU). A,B, The ROI was placed if multiple layers were recognized and the layers were thick enough to evaluate HUs accurately. The largest circular ROIs were used to measure the HU of the thick middle/outer layer without affecting adjacent layers. For each dog, HU was measured three times, and the average HUs were calculated. C, The gastric wall thickness was measured perpendicular to the wall at the thickest region avoiding rugal folds. 100 kVp, 250 mA, soft tissue algorithm, and a slice thickness of 1.25 mm were used

thickening was further categorized to greater or lesser curvature if the thickening was in the gastric body. The gastric wall thickness was measured perpendicular to the wall at the thickest region avoiding rugal folds.

3 | RESULTS

3.1 | Clinical findings

3.1.1 | Group I

Three dogs underwent necropsy within 24 h of the CT study and were confirmed to have gastric wall edema within the submucosal layer. The clinical diagnoses for the three dogs included peritonitis, coagulopathy, and cerebral hemorrhage. All three dogs had blood sample collection within 24 h from the time of CT study, and one dog had hypoalbuminemia (<2.3 g/dL).

3.1.2 | Group II

Thirteen dogs had pre- and postcontrast abdominal CT and gastric wall edema listed as one of the differential diagnoses in CT reports. Two dogs were excluded from the present study due to histologic confirmation of gastric wall neoplasia. Two dogs with a two-layer appearance and enhancing soft tissue attenuating outer layer were excluded since three-layer appearance in the postcontrast study with non-enhancing, thick, and fluid attenuating middle layer was considered to be one of the important CT features of gastric wall edema in group I. After exclusion, CT studies of nine dogs were reviewed in the present study.

In group II, clinical diagnoses possibly related to gastric wall thickening were present in eight/nine dogs: hypoalbuminemia (5) [[protein-losing enteropathy (PLE) (2) or nephropathy (PLN) (1), hypoalbuminemia from other causes (2)], pancreatitis (1), gastric ulcer (1), and five days post-gastropexy (1). One dog did not have a clinical diagnosis likely to be a cause of gastric wall thickening, and the dog was diagnosed with urinary obstruction. Seven/nine dogs had blood sample collection within 24 h from the time of CT study, and six/nine dogs had hypoalbuminemia (<2.3 g/dL).

3.2 | CT acquisition technical parameters

All CT studies were performed using a 64-slice multidetector CT machine (Light Speed VCT, GE Medical Systems Inc., Waukesha, WI) with images acquired in the transverse plane using the following image acquisition parameters: helical scan mode, 100–120 kVp, 240–340 mA, slice thickness = 0.625–2.5 mm, tube rotation time = 1 s, pitch = 1, matrix = 512×512, and detail algorithm. Postcontrast images were acquired 80–95 s following the start of intravenous administration of nonionic iodinated contrast agent (2 mL/kg, iohexol, Omnipaque™ 240, GE Healthcare, Malborough, MA, USA). The contrast dose was administered using either manual or a power injector (Medrad Mark V ProVis, Indianola, PA, USA). The injection rate was adjusted such that the total volume of contrast was delivered over 20 s. Dogs were positioned in sternal recumbency under sedation or general anesthesia.

3.3 | CT characteristics of confirmed gastric wall edema in dogs (Group I)

Two/three dogs with confirmed gastric wall edema had pre- and postcontrast CT studies of the stomach, and one dog only had a postcontrast CT study of the stomach. Computed tomographic findings and clinical diagnoses for individual dogs in group I were provided in Table S1.

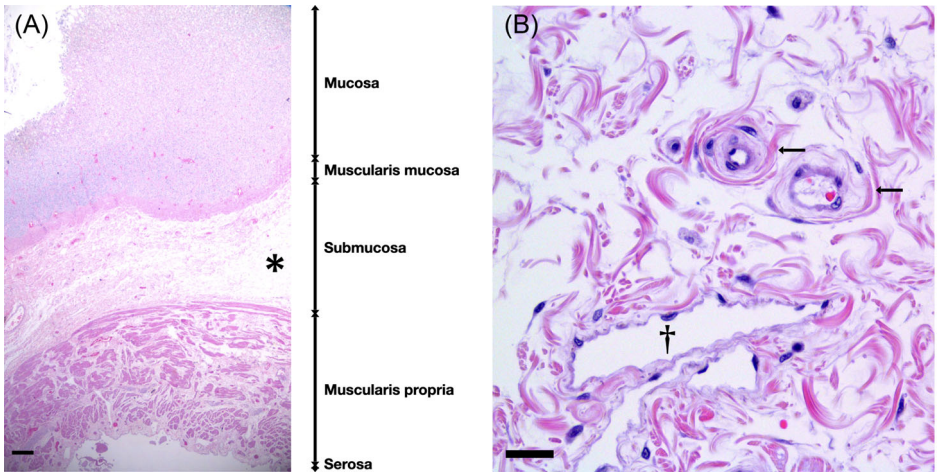

FIGURE 2 Photomicrograph of gastric submucosal edema in a dog: low magnification image (A) and close-up image of submucosal layer (B). A, There is a diffuse extensive edema in the submucosal layer with increased spacing and separation of collagen fibers caused by a nonstaining fluid within gastric submucosa (asterisk). 20× magnification, Bar = 200 um. B, Perivascular edema is present with expanding adventitia by a nonstaining fluid surrounding the vessel (black arrow). Lymphatics were dilated (dagger). Hematoxylin and eosin. 40× magnification, Bar = 25 um

In pre-contrast CT studies, a three-layer appearance of the gastric wall was present with a faintly recognizable hypoattenuating middle layer and ill-defined hyperattenuating inner and outer layers in one dog, and a single hypoattenuating layer in the other dog. In post-contrast CT studies, the well-defined three-layer appearance was present in all three dogs due to strong enhancement of the inner (mucosal) and mild enhancement of the outer layer (muscularis/serosal). The middle layer was thick and fluid attenuating with no contrast enhancement in all three dogs (average HU of 18.2 [14.3–22.0] in precontrast and 24.7 [19.8–31.2] in postcontrast studies). It was not possible to accurately place ROIs in the thin inner layer and thin outer layers when three layers were present due to small regions and partial volume averaging artifact. Thin enhancing blood vessels in the thick middle layer were present in the postcontrast CT studies in two dogs.

The average thickest gastric wall thickness was 11.1 mm (8.6-12.9 mm) in three dogs. A dog with cerebral hemorrhage showed diffuse gastric wall thickening, with fundus being the thickest, up to 11.8 mm in thickness. The remaining two dogs showed focal asymmetric thickening of the gastric wall. One dog with fibrinosuppurative peritonitis showed multifocal thickening involving the fundus, the greater curvature of the body, and the pylorus, with the greater curvature being thickest, up to 12.9 mm in thickness. The other dog with coagulopathy showed focal thickening in the pylorus, up to 8.6 mm. In all three dogs, histopathology confirmed the presence of edema in the submucosa (Figure 2).

3.4 | CT characteristics of presumed gastric wall edema in dogs (Group II)

All nine dogs with presumed gastric wall edema had both pre- and postcontrast CT studies of the stomach.

In pre-contrast studies, eight dogs had a well-defined three-layer appearance with fluid attenuating middle layer and hyperattenuating inner and outer layers (Figure 3A). A single-layer gastric wall with fluid attenuation was present in one dog in the precontrast study.

In postcontrast CT studies, nine dogs showed a distinct three-layer appearance with the thin strongly enhancing inner layer, non-enhancing fluid attenuating middle layer (average HU of 14.1 [6.9–21.3] in precontrast and 19.0 [8.7–29.3] in postcontrast studies), and thin mildly enhancing outer layer (Figures 3B,C, 4B, and 5B).

It was not possible to accurately place ROIs in the thin inner layer and thin outer layers when three-layer appearance waspresent due to small regions and partial volume averaging artifact.

Thin tortuous and tubular contrast-enhancing blood vessels were present through the gastric wall in all dogs in the postcontrast CT studies (Figure 6).

In these nine dogs, one dog showed diffuse gastric wall thickening (Figure 3), six dogs had focal concentric gastric wall thickening (Figure 4), and two dogs had focal asymmetric thickening (Figure 5). In these eight dogs with focal gastric wall thickening, the gastric wall thickening was most commonly involving the pyloric part (7/8), followed by the body (4/8). None of the dogs with focal gastric wall thickening had thickening of the gastric fundus. Three dogs had continuous gastric wall thickening through the gastric body and pylorus. No dogs had multifocal gastric wall thickening. The average gastric wall thickness of the thickened three-layer appearance wall was 12.2 ± 4.3 mm.

When we combined dogs from groups I and II, diffuse gastric wall thickening was present in one dog with hypoalbuminemia (Figure 3) and the other dog with cerebral hemorrhage. Focal concentric gastric wall thickening was present in five dogs with hypoalbuminemia (Figure 4) and one dog with urinary obstruction. Focal asymmetric gastric wall thickening involving fundus, body, and pylorus was present in one dog with peritonitis and concomitant hypoalbuminemia. Focal

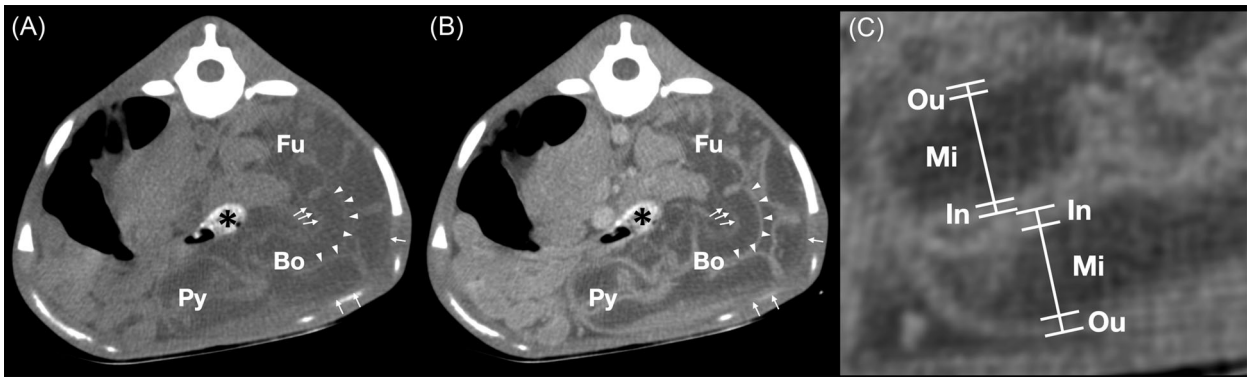

FIGURE 3 Transverse images of pre- (A) and post-contrast (B) CT of gastric wall edema and magnified image of the pyloric gastric wall (C) in a dog with hypoalbuminemia in soft tissue window (window level: 40 HU, window width: 350 HU). Gastric wall is diffusely thickened with three-layer appearance characterized by a thin inner and outer enhancing soft tissue attenuating layer and thick non-enhancing fluid attenuating middle layer. Arrowheads and arrows showing the luminal and serosal margins of the gastric wall, respectively. The stomach is filled with small amount of fluid. A small amount of heterogeneously mineral attenuating fecal material is present in the transverse colon (Black asterisk). Fu, Gastric fundus: Bo, Gastric body: Py, Pyloric part: In, inner layer: Mi, middle layer: Ou, outer layer. 100 kVp, 250 mA, soft tissue algorithm and a slice thickness of 1.25 mm were used

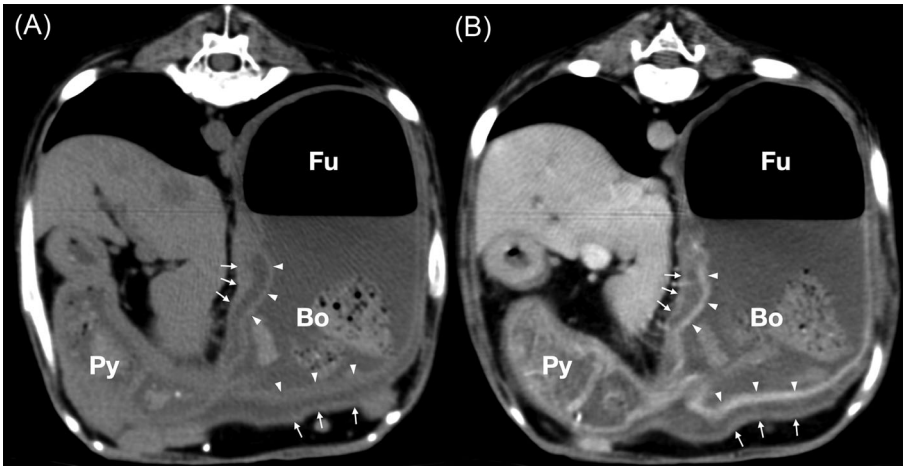

FIGURE 4 Transverse images of pre- (A) and postcontrast (B) CT of the gastric wall edema in a dog with hypoalbuminemia in soft tissue window (window level: 40 HU, window width: 350 HU). Focal concentric gastric wall thickening with three-layer appearance (a thin inner and outer enhancing soft tissue attenuating layer and thick non-enhancing fluid attenuating middle layer) is present in the gastric body. The gastric wall of the fundus is normal and thin. Arrowheads and arrows showing the luminal and serosal margins of the gastric wall, respectively. The stomach is filled with fluid, gas, and heterogeneous soft tissue attenuating materials. 100 kVp, 300 mA, soft tissue algorithm and a slice thickness of 1.25 mm were used. Fu, Gastric fundus: Bo, Gastric body: Py, Pyloric part

thickening involving only the gastric pylorus in three dogs was present with gastric ulcer, pancreatitis (Figure 5), or coagulopathy. The focal asymmetric gastric wall thickening in the dog with pancreatitis was adjacent to the pancreas with changes consistent with pancreatitis.

4 | DISCUSSION

Based on our review of the literature, this is the first published report describing the CT features of histologically confirmed gastric wall edema in dogs. The predominant CT features in these three dogs were a three-layer appearance with gastric wall thickening, a thin inner and outer enhancing soft tissue attenuating layer, and a thick non-enhancing fluid attenuating middle layer with the presence of conspicuous small blood vessels in postcontrast CT studies.

In humans, the CT features of GI wall edema have been reported as a two- or three-layer appearance of the GI wall with a poorly enhancing middle layer. However, seen more commonly in the colon and the small intestine than in the stomach.[17] Although gastric wall thickening with a concentric two-layer appearance is also considered as gastric wall edema in humans: these two layers are the inner hypoattenuating layer and outer hyperattenuating layer.[9,10] This two-layer appearance with gastric wall edema in humans is considered to be secondary to mucosal damage such as gastric ulcer as a cause of gastritis and gastric wall edema or the quality of the CT examination such as inadequate contrast administration or partial volume averaging artifact.[9] In the

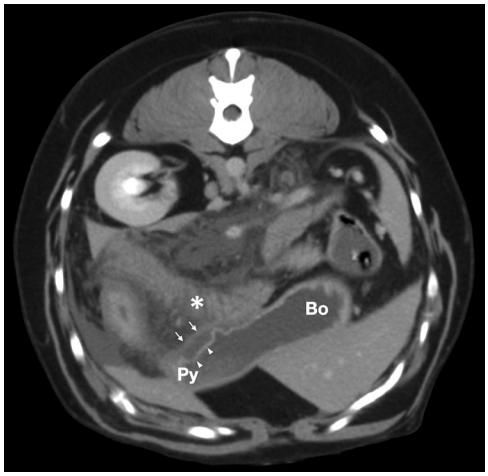

FIGURE 5 The postcontrast CT image using multiplanar reconstruction in a dog with pancreatitis in soft tissue window (window level: 40 HU, window width: 350 HU). Focal asymmetric gastric wall thickening with three-layer appearance (a thin inner and outer enhancing soft tissue attenuating layer and thick non-enhancing fluid attenuating middle layer) was present at the pyloric part of the stomach adjacent to the pancreas (white asterisk). Arrowheads and arrows showing the luminal and serosal margins of the gastric wall, respectively. The pancreas was diffusely contrast enhancing with fluid attenuation between the pancreatic lobes indicating pancreatic edema. Peripancreatic steatitis was present as wispy fluid attenuation surrounding the pancreas. The stomach is filled with fluid attenuation and a small amount of heterogeneous soft tissue attenuating material. 140 kVp, 300 mA, soft tissue algorithm and a slice thickness of 3.75 mm were used. Bo, Gastric body: Py, Pyloric part

present study, two dogs who were excluded from the group II also had a two-layer appearance in the post-contrast studies, however, these two layers were a thin enhancing inner layer and mildly enhancing outer soft tissue attenuating layer, which is a normal enhancement pattern of the canine stomach.[1,8]

There was no previous publication reporting HU of the poorly enhancing middle layer in the CT images of gastric wall edema in humans or veterinary medicine, based on our review of the literature. In humans, the possible diagnosis with a similar hypoattenuating thickened submucosal layer is fat deposition. However, all cases seen in the present study were not diagnosed as fat deposition as the measured HU did not correspond to fat (negative value). Few veterinary articles reported ultrasonographic features of gastric wall edema, and the distinct features were thickening of the submucosal layer alone or thickening of submucosal and muscularis layers.[18–20] The thick middle layer seen in the present study is most likely the submucosal layer since the thickening of the submucosal layer was histologically observed in dogs in Group I. However, involvement of the muscularis layer is also possible, as it is impossible to differentiate each individual layer of the gastric wall on CT, except the strong contrast-enhanced mucosal layer.[8] The most outer contrast enhancing layer is probably the serosal or the muscularis layer, based on the human CT features of gastric wall edema.[3,10,21] In humans, the thick hypoattenuating inner or middle layer of the stomach is considered the submucosal layer.[21]

Although the three-layer appearance was visible in most of the dogs in the pre-contrast studies, the distinct three-layer appearance was more conspicuous in the post-contrast studies due to the enhancement of inner and outer layers contradict relative to the non-enhancing middle layer. Small vessels were visible in the entire gastric wall, but were more conspicuous when a thickened, poorly enhancing middle layer was present (Figure 6).

Gastric wall edema causes gastric wall thickening and is non-specific as it is known to occur secondary to multiple disease processes; including hypoalbuminemia, inflammation, or neoplasia. However, the gastric wall edema itself is a benign lesion, and it is important to differentiate it from primary malignant gastric wall thickening such as neoplasia. Generally, gastric neoplasias have CT features of homogeneously or heterogeneously contrast-enhancing mass in the gastric wall with loss of layering or focal enhancing mucosal mass with a preserved outer

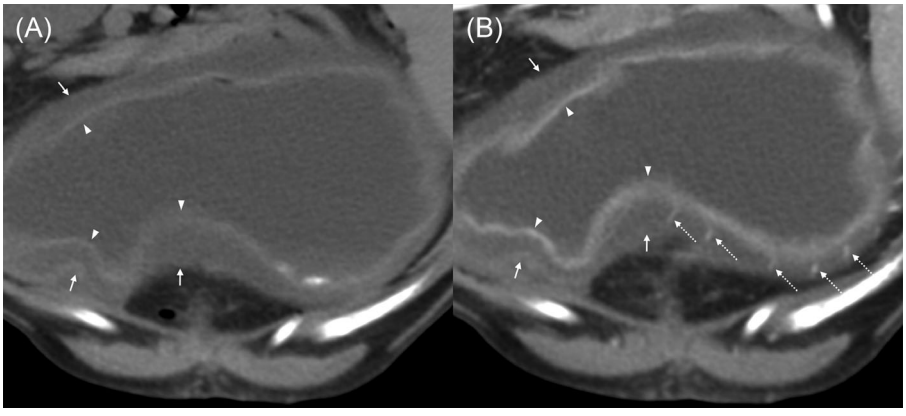

FIGURE 6 Transverse images of pre- (A) and post-contrast (B) CT of the gastric wall edema in a dog in soft tissue window (window level: 40 HU, window width: 350 HU). Focal concentric gastric wall thickening of the gastric body with three-layer appearance (a thin inner and outer enhancing soft tissue attenuating layer and thick non-enhancing fluid attenuating middle layer) is present. Multiple small tortuous contrast enhancing blood vessels are present in the fluid attenuating middle layer of the gastric wall (dotted arrows). Arrowheads and arrows showing the luminal and serosal margins of the gastric wall, respectively. The stomach is filled with fluid. 140 kVp, 340 mA, soft tissue algorithm, and a slice thickness of 3.75 mm were used

less enhancing layer in humans.[22] There is no detailed description of whether the mucosal enhancement and layering were preserved or not in the CT studies of gastric tumor in dogs.[23]

Reported causes of the gastric wall edema in dogs include inflammatory diseases such as gastritis with or without ulceration,[24,25] uremic gastropathy,[26] inflammation adjacent to the stomach such as pancreatitis,[19] gastric outflow obstruction with gastric dilatation and volvulus[27,28] or pylorogastric or duodenogastric intussusception,[20,29] gastric neoplasia,[30,31] portal hypertension,[11] and hypoalbuminemia.[18] In the present study, gastric wall edema was present in similar reported diseases: however, more commonly seen in dogs with hypoalbuminemia.

Of seven dogs with hypoalbuminemia, six dogs had diffuse or concentric gastric wall thickening, and one dog had focal asymmetric gastric wall thickening with concomitant peritonitis. Concentric gastric wall thickening was present involving the body and the pylorus of the stomach. This was similar to the ultrasonographic findings of generalized gastric wall edema seen in dogs with hypoalbuminemia.[18] There is a limitation of gastric wall evaluation in ultrasound due to artifacts caused by intraluminal gas and materials leading to an incomplete evaluation of the dorsal gastric wall. The CT enabled us to evaluate the entire gastric wall without artifact. In the present study, the gastric wall edema secondary to hypoalbuminemia was more commonly present in the gastric body and the pylorus and not the gastric fundus.

The thickness of gastric wall edema has not been previously reported. The average thickness of the gastric wall edema in the present study (12.2±4.3 mm) was considered moderately thicker than the reported normal gastric wall thickness (up to 4.9 mm).[1] However, the thicknesses of some gastric wall neoplasia also can be around 10 mm.[1] Thus, gastric wall edema cannot be differentiated from malignant disease based on the gastric wall thickness alone.

The focal gastric wall edema was reported adjacent to the site of pancreatitis in dogs using ultrasound,[19] and adjacent to the site of the gastric mucosal defect in humans using CT.[32] In humans, there is no known correlation between the location of gastric wall edema and pancreatitis. Similar focal asymmetric thickenings of the gastric wall adjacent to the region of pancreatitis or ulceration were observed in the present study.

Direct causes of the gastric wall edema were not identified in two dogs, one with urinary obstruction and the other with cerebral hemorrhage. In the dog with urinary obstruction, serum albumin level measured three days prior to the CT study was at the lower end of the normal reference range, 2.3 g/dL, and the serum albumin level was not evaluated within 24 h from the CT study. Thus, the dog may have developed hypoalbuminemia and was not documented during the 24 h of the CT study. The uremia was also considered as the cause of gastric wall edema, however, the urinary obstruction was resolved by placing urinary catheter and azotemia has already been resolved at the day of the CT study. The dog with cerebral hemorrhage in the present study also did not have serum albumin level evaluation within 24 hours from the CT study. In humans, it is known that traumatic brain injury can cause GI dysfunction and secondary inflammation of the GI tract.[33,34] It is possible that the dog with cerebral hemorrhage had concomitant gastric dysfunction leading to gastritis because the stomach in the dog with cerebral hemorrhage was markedly distended and filled with fluid showing evidence of functional ileus.

Limitations of the present study were the retrospective nature of the study and only a small number of histopathologically confirmed dogs. The small number of dogs with histopathologic confirmation was primarily due to the fact that gastric wall edema is usually a transient phenomenon and edema itself is not fatal condition. Also, histopathological information was provided only from histopathology report and slides were not re-reviewed for the current study.

In conclusion, the CT features identified in this sample of dogs with gastric wall edema included the following: thickened gastric wall with three-layer (thin inner and outer enhancing soft tissue attenuating layers and thick non-enhancing fluid attenuating middle layer) appearance in both pre- and the postcontrast CT studies and presence of thin enhancing blood vessels in the thick middle layer in the post-contrast CT studies. The most common clinical finding was hypoalbuminemia. An improved awareness of the CT features of gastric wall edema in dogs can be used to help clinicians differentiate this benign condition from gastric neoplasia. Future prospective studies with detailed histopathological evaluations immediately after CT would be needed to better understand the underlying pathophysiology and extent of involvement for each of the gastric wall layers.

LIST OF AUTHOR CONTRIBUTIONS

Category 1

(a) Conception and Design: Murakami, Heng
(b) Acquisition of data: Murakami, Heng, Sola
(c) Analysis and Interpretation of Data: Murakami, Heng, Sola

Category 2

(a) Drafting the Article: Murakami
(b) Revising the Article: Murakami, Heng, Sola

Category 3

(a) Final Approval: Murakami, Heng, Sola

Category 4

(a) Agreement to be accountable for all aspects of the work ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved: Murakami, Heng, Sola

CONFLICT OF INTEREST

The authors have declared no conflict of interest.

PREVIOUS PRESENTATION DISCLOSURE

Presented in as an oral presentation at the 2020 Annual Scientific Conference of the American College of Veterinary Radiology.

EQUATOR NETWORK DISCLOSURE

An EQUATOR network checklist was not used.

ORCID

Masahiro Murakami https://orcid.org/0000-0001-7816-8311
Hock Gan Heng https://orcid.org/0000-0001-5302-5402

REFERENCES

1. Hoey S, Drees R, Hetzel S. Evaluation of the gastrointestinal tract in dogs using computed tomography. *Vet Radiol Ultrasound*. 2013;54:25-30.
2. Terragni R, Vignoli M, Rossi F, et al. Stomach wall evaluation using helical hydro-computed tomography. *Vet Radiol Ultrasound*. 2012;53:402-405.
3. Fernandes T, Oliveira MI, Castro R, Araujo B, Viamonte B, Cunha R. Bowel wall thickening at CT: simplifying the diagnosis. *Insights Imaging*. 2014;5:195-208.
4. Chokkappan K, Chinchure D, Chawla A, et al. An approach to various gastric pathologies using an "image appearance-based classification" on computed tomography. *Curr Probl Diagn Radiol*. 2016;45:23-38.
5. Pfau PR, Perlman SB, Stanko P, et al. The role and clinical value of EUS in a multimodality esophageal carcinoma staging program with CT and positron emission tomography. *Gastrointest Endosc*. 2007;65:377-384.
6. Bhandari S, Shim CS, Kim JH, et al. Usefulness of three-dimensional, multidetector row CT (virtual gastroscopy and multiplanar reconstruction) in the evaluation of gastric cancer: a comparison with conventional endoscopy, EUS, and histopathology. *Gastrointest Endosc*. 2004;59:619-626.
7. Kumano S, Murakami T, Kim T, et al. T staging of gastric cancer: role of multi-detector row CT. *Radiology*. 2005;237:961-966.
8. Fitzgerald E, Lam R, Drees R. Improving conspicuity of the canine gastrointestinal wall using dual phase contrast-enhanced computed tomography: a retrospective cross-sectional study. *Vet Radiol Ultrasound*. 2017;58:151-162.
9. Chou CK, Wu RH, Mak CW, Lin MP. Clinical significance of poor CT enhancement of the thickened small-bowel wall in patients with acute abdominal pain. *AJR Am J Roentgenol*. 2006;186:491-498.
10. Macari M, Balthazar EJ. CT of bowel wall thickening: significance and pitfalls of interpretation. *AJR Am J Roentgenol*. 2001;176:1105-1116.
11. Specchi S, Rossi F, Weisse C, et al. Canine and feline abdominal arterioportal communications can be classified based on branching patterns in computed tomographic angiography. *Vet Radiol Ultrasound*. 2018;59:687-696.
12. Bertolini G. Acquired portal collateral circulation in the dog and cat. *Vet Radiol Ultrasound*. 2010;51:25-33.
13. Agnello F, Albano D, Micci G, et al. CT and MR imaging of cystic renal lesions. *Insights Imaging*. 2020;11:5.
14. Galia M, Albano D, Bruno A, et al. Imaging features of solid renal masses. *Br J Radiol*. 2017;90:20170077.
15. Benjaminov O, Atri M, O'Malley M, Lobo K, Tomlinson G. Enhancing component on CT to predict malignancy in cystic renal masses and interobserver agreement of different CT features. *AJR Am J Roentgenol*. 2006;186:665-672.
16. Evans HE, Miller ME. *Miller's Anatomy of The Dog*. Elsevier; 2013;
17. Balthazar EJ. CT of the gastrointestinal tract: principles and interpretation. *AJR Am J Roentgenol*. 1991;156:23-32.
18. Murakami M, Heng HG, Lim CK, Parnell NK, Sola M. Ultrasonographic features and prevalence of presumed gastric wall edema in dogs with hypoalbuminemia. *J Vet Intern Med*. 2020;34:1867-1871.
19. Murakami M, Heng HG, Lim CK, et al. Ultrasonographic features of presumed gastric wall edema in 14 dogs with pancreatitis. *J Vet Intern Med*. 2019;33:1260-1265.
20. Choi J, Keh S, Kim T, Jang J, Kim H, Yoon J. Ultrasonographic findings of pylorogastric intussusceptions in two dogs. *J Vet Sci*. 2012;13:215-217.
21. Walter J, Ayoob A, DiSantis D. The bowel wall target sign. *Abdom Imaging*. 2015;40:457-458.
22. Hargunani R, Maclachlan J, Kaniyur S, Power N, Pereira SP, Malhotra A. Cross-sectional imaging of gastric neoplasia. *Clin Radiol*. 2009;64:420-429.
23. Tanaka T, Akiyoshi H, Mie K, Okamoto M, Yoshida Y, Kurokawa S. Contrast-enhanced computed tomography may be helpful for characterizing and staging canine gastric tumors. *Vet Radiol Ultrasound*. 2019;60:7-18.
24. Penninck D, Matz M, Tidwell A. Ultrasonography of gastric ulceration in the dog. *Vet Radiol Ultrasound*. 1997;38:308-312.
25. Suarez-Esquivel M, Alfaro-Alarcon A, Guzman-Verri C, Barquero-Calvo E. Analysis of the association between density of Helicobacter spp and gastric lesions in dogs. *Am J Vet Res*. 2017;78:1414-1420.
26. Grooters AM, Miyabayashi T, Biller DS, Merryman J. Sonographic appearance of uremic gastropathy in four dogs. *Veterinary Radiology & Ultrasound*. 1994;35:35-40.
27. Lantz GC, Bottoms GD, Carlton WW, Newman S, Cantwell HD. The effect of 360 gastric volvulus on the blood supply of the nondistended normal dog stomach. *Vet Surg*. 1984;13:189-196.
28. Sharp CR, Rozanski EA. Cardiovascular and systemic effects of gastric dilatation and volvulus in dogs. *Topics Compan Anim Med*. 2014;29:67-70.
29. Allman DA, Pastori MP. Duodenogastric intussusception with concurrent gastric foreign body in a dog: a case report and literature review. *J Am Anim Hosp Assoc*. 2013;49:64-69.
30. Patnaik AK, Hurvitz AI, Johnson GF. Canine gastric adenocarcinoma. *Vet Pathol*. 1978;15:600-607.
31. Terragni R, Vignoli M, van Bree HJ, Gaschen L, Saunders JH. Diagnostic imaging and endoscopic finding in dogs and cats with gastric tumors: a review. *Schweiz Arch Tierheilkd*. 2014;156:569-576.
32. Oguro S, Funabiki T, Hosoda K, et al. 64-Slice multidetector computed tomography evaluation of gastrointestinal tract perforation site: detectability of direct findings in upper and lower GI tract. *Eur Radiol*. 2010;20:1396-1403.
33. Baguley IJ, Heriseanu RE, Cameron ID, Nott MT, Slewa-Younan S. A critical review of the pathophysiology of dysautonomia following traumatic brain injury. *Neurocrit Care*. 2008;8:293-300.
34. Hang CH, Shi JX, Li JS, Wu W, Yin HX. Alterations of intestinal mucosa structure and barrier function following traumatic brain injury in rats. *World J Gastroenterol*. 2003;9:2776-2781.

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Murakami M, Heng HG, Sola M. CT features of confirmed and presumed gastric wall edema in dogs. *Vet Radiol Ultrasound*. 2022;63:711–718. https://doi.org/10.1111/vru.13123