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# Comprehensive evaluation and management of chronic gastritis in a dog: A case report

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**Abstract**

*Gastric ulcer is a significant clinical issue in dogs, with higher prevalence in mature animals and often occurs secondary to systemic diseases such as hepatic or renal disorders, Addison's disease or shock. This case report highlights a four-and-a-half-year-old intact male Rottweiler presented with persistent vomiting, anorexia, abdominal pain and anemia. Diagnostic imaging, including radiography, ultrasonography and endoscopy, revealed gastric ulceration and the presence of radiopaque lead pellets in the pyloric region of the stomach. Histopathological examination confirmed extensive gastric mucosal damage. Treatment included the elimination of lead, fluid therapy and administration of proton pump inhibitors, antibiotics and mucosal protectants. Incorporating psyllium husk into the diet helped to enhance lead excretion. Follow-up imaging after two weeks showed absence of lead pellets and normal gastric mucosa. This case underscores the importance of comprehensive diagnostic and therapeutic approaches for chronic gastric ulcers in dogs, emphasising the role of multimodal imaging and targeted treatment strategies.*

**Keywords:** *Dog, gastric ulcers, diagnostics, therapeutics*

Gastric ulcers are prevalent in both young and adult dogs, with a higher incidence in mature animals. These ulcers often arise secondary to systemic diseases such as hepatic or renal disorders, Addison's disease, or shock (Hall, 2000). Clinical presentation of gastric ulcers in dogs may include acute abdominal signs such as pain, distension or vomiting, or more subtle, non-specific signs like lethargy, inappetence and weakness (Stanton and Bright, 1989).

The gastric epithelial layer undergoes continuous renewal, with surface mucous cells replaced every three days and deeper neck mucous cells renewed approximately every seven days (Strombeck and Guilford, 1990). Gastric ulcers can result from physical damage, such as from a foreign body and chemical alterations like exposure to hydrochloric acid (HCl), bile acids and pancreatic enzymes. Disruption of the epithelial barrier allows HCl to diffuse back across the mucosa, stimulating the intrinsic nervous system and causing further HCl and pepsin release. Hydrochloric acid can also directly activate tissue mast cells, leading to increased histamine release and subsequent mucosal damage. Exogenous toxins, such as lead, may further exacerbate gastric mucosal injury (Otto *et al*., 1991).

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Imaging modalities play a crucial role in the diagnosis and management of gastric ulcers. Ultrasound and endoscopy are complementary techniques providing valuable insights into gastrointestinal (GI) health. Although contrast radiography has traditionally been used to detect gastric ulcers (Terragni *et al*., 2014), its use has declined with the advent of ultrasonography. Ultrasonographic detection of gastric ulcers often reveals a mucosal defect with a thickened gastric or intestinal wall, with a collection of small echoes, likely representing gas bubbles (Paoloni *et al*., 2002). Endoscopy, being minimally invasive, serves both as a diagnostic tool and for therapeutic interventions, such as removing foreign bodies. Although computed tomography (CT) is well-established for investigating GI bleeding in humans (Soto *et al*., 2015), its use in canine GI ulceration remains under-reported.

Therapeutic management of gastric ulcers involves identifying and addressing potential causes, such as lead toxicity, and employing treatments to prevent secondary bacterial infections. Therapeutic options include antibiotics, antacids, H2-receptor blocker, proton pump inhibitors, and mucosal protectants like sucralfate. This case report presents comprehensive diagnostic and therapeutic strategies for managing chronic gastric ulcers in a dog.

A four-and-a-half-year-old intact male Rottweiler presented with persistent vomiting characterized by foamy yellowish vomitus. Based on the owner's account, it was suspected that the dog had accidentally ingested lead gun pellets used for recreational hunting. The dog had exhibited signs of anorexia, weight loss, abdominal pain and lethargy over the past week. The case was previously treated by a local veterinarian with oral antiemetics, but there were no signs of improvement. During the physical examination, the patient's vital signs were recorded as follows: a temperature of 101.7°F, heart rate of 120 beats per minute, and respiratory rate of 30 breaths per minute. Additionally, the patient exhibited signs of anaemia, with pale mucous membranes. Abdominal palpation revealed tenderness and discomfort in the abdominal region. Haematological and biochemical blood tests indicated reduced haemoglobin levels (7.5 g/dL) and a low red blood cell count (4.5 x$10^6$/µL), while all other biochemical parameters were within the normal range.

Diagnostic tests, including imaging (ultrasound and radiography) and endoscopy, were conducted to determine the precise cause and progression of the disease. Radiography and ultrasonography were performed to evaluate the structural integrity of the gastric wall and to detect abnormalities such as wall thickening, perforation, fluid accumulation, foreign bodies, neoplastic conditions, volvulus and strictures. Radiography revealed radiopaque lead pellets, indicated by metallic opacity in the pyloric region of the stomach (Fig. 1).

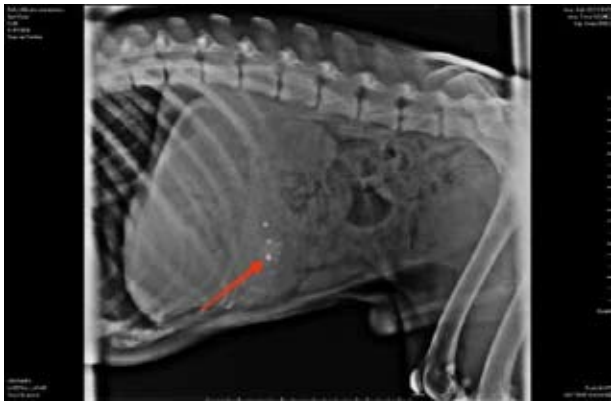

*Fig. 1:* Abdominal radiograph showing metallic opacity (red arrow) in the pyloric region.

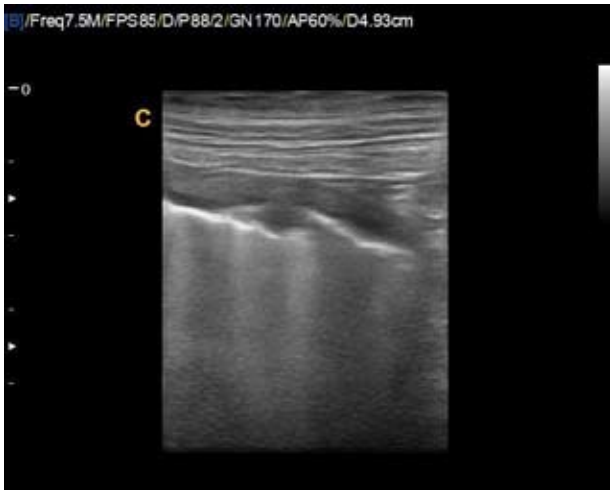


*Fig. 2:* Sonogram revealing a thickened and hypoechoic gastric wall with a crater-shaped ulcer in the stomach.

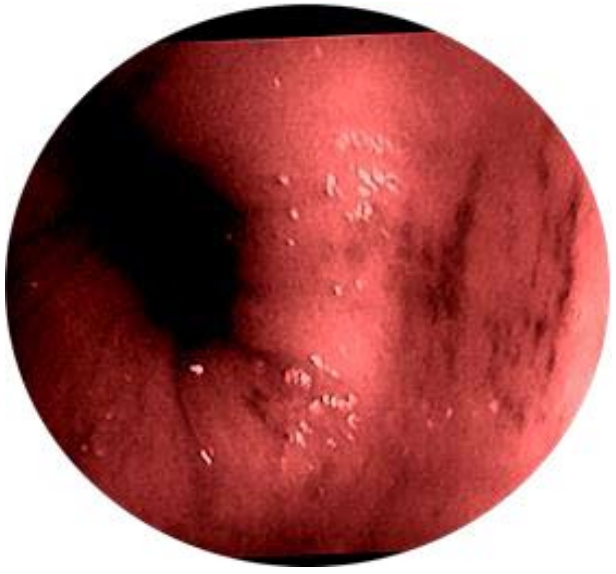

**Fig. 3:** Endoscopic view showing mucosal erythema at the cardia of the stomach.

To assess the structural integrity of the gastric wall and identify abnormalities such as thickening or fluid accumulation, an ultrasound scanning was conducted. Food was withheld for 12 hours prior to the sonographic

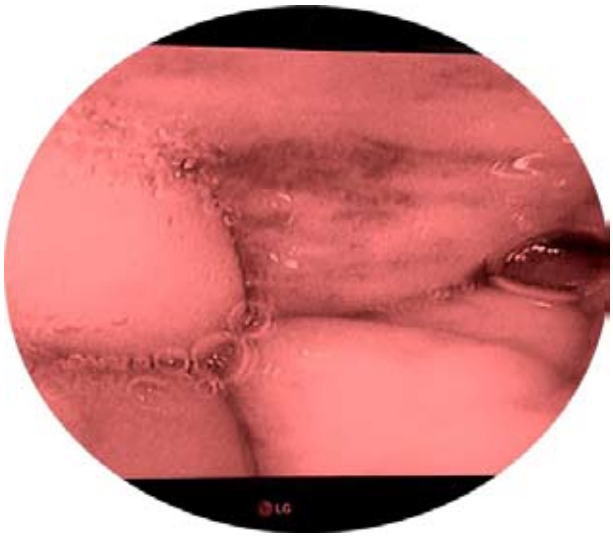

*Fig. 4:* Endoscopic appearance of gastric mucosal erythema with excess froth

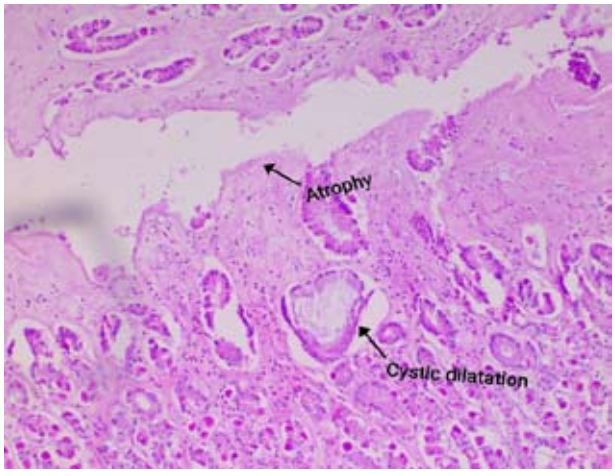


*Fig. 5:* Histopathological slide demonstrating gastric glandular atrophy and cystic dilation.

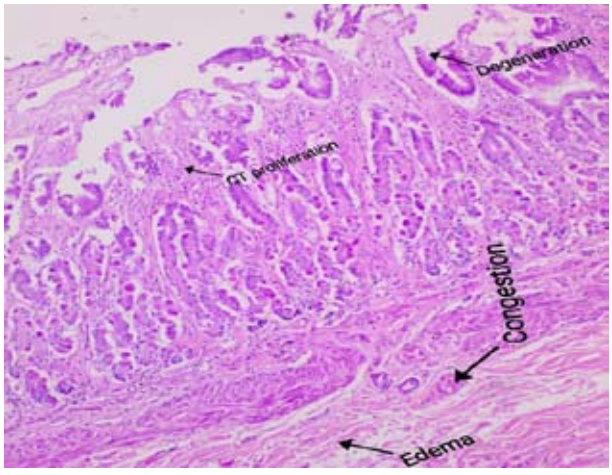


**Fig. 6:** Histopathological slide showing glandular degeneration with submucosal edema.

evaluation. The hair on the ventral abdomen was shaved, and coupling gel was applied. The examination was carried out in lateral recumbency using a curved array transducer with frequencies ranging from 8 to 10 MHz. The ultrasound findings showed alterations in the gastric mucosa, including a thickened, hypoechoic gastric wall and a crater-shaped ulcer in the stomach (Fig. 2).

For the identification of lesions in the GIT and collection of biopsy samples, endoscopy was performed. The dog was fasted for 24 hours for solids and 6 hours for water before the endoscopic examination. A flexible video endoscope (Karl Storz, AIDA™ Compact II, Germany) with a working length of 140 cm, an outer diameter of 9 mm, an accessory/biopsy channel diameter of 2.8 mm, and a four-way deflection knob was used. Endoscopy was performed under general anesthesia. Premedication was administered using atropine at 0.04 mg/kg intramuscularly 20 minutes prior to induction to decrease gastric secretions and xylazine at 1.5 mg/kg intramuscularly. An intra-venous catheter was placed and a balanced electrolyte solution (Lactated Ringer's) was administered at 5 ml/kg/h during anesthesia. Anesthesia was induced with an intramuscular injection of ketamine at 7.5 mg/kg and maintained using a combination of ketamine and diazepam (1:25 w/v). The dog was intubated and a mouth gag was placed before the examination. Endoscopic examination revealed mucosal erythema and ulceration at the cardia of the stomach (Fig. 3, 4).

Gastric mucosa samples were obtained from the edge of the ulcer using endoscope-guided "alligator jaw" biopsy forceps for histopathological analysis. The samples were fixed in a 10 per cent buffered formalin solution. After fixation, the samples were embedded in paraffin, sectioned at 4 µm, and stained with hematoxylin and eosin for histological evaluation. The histopathological examination showed significant architectural distortion, with notable gastric glandular atrophy and cystic dilation (Fig. 5, 6).

On the basis of anamnesis and clinical findings, the case was diagnosed as chronic gastric ulceration due to lead pellet ingestion. Following the diagnosis, therapeutic intervention was initiated. The owner was advised to withhold food for three days to allow the vomiting to subside and to minimise gastric acid secretion. During this period, the dog was maintained on fluid therapy with 300 ml of normal saline (NS) and 200 ml of lactated Ringer's (RL) administered intravenously. The patient received pantoprazole at 1 mg/kg IV once daily, amoxicillin at 10 mg/kg IM twice daily and ranitidine at 2 mg/kg SC twice daily for seven days. A soluble fibre, psyllium husk, was added to the diet at a dose of five tablespoons per day for one month to help eliminate the lead pellets and reduce the pathological signs. Supportive treatment included omeprazole at 1 mg/kg orally once daily and sucralfate at one gram orally twice daily, was continued for one month. The owner was advised to keep a log of the vomiting and faecal characteristics, including type, frequency and colour. Two weeks after presentation, the patient was subjected to diagnostic ultrasound and radiography, revealing the

absence of lead pellets and a normal gastric mucosa. One month later, the dog had an uneventful recovery with a normal appetite.

Gastric ulcers form when the gastric mucosal cytoprotective barrier is compromised by "aggressive factors" such as hydrochloric acid, pepsin, trypsin, stimulants of hydrochloric acid secretion, oxygen free radicals, drugs (particularly NSAIDs), platelet-activating factors and bile acids (Kubiak *et al*., 2004; Parrah *et al*., 2013; Tolbert and Gould, 2020). Ulcers develop when the equilibrium between mucosal defense mechanisms and ulcerogenic factors, such as acid and pepsin, is disrupted. Common predisposing factors include NSAID use, gastrointestinal neoplasia or hepatobiliary disease (Hinton *et al*., 2002; Cariou *et al*., 2009). However, none of these factors were identified in the current case.

Lead toxicity can cause continuous irritation and physical damage to the gastric mucosa, impairing mucosal defense mechanisms. Sass (1970) first documented the association between lead poisoning in dogs and perforating gastrointestinal ulcers. Berny *et al*. (1992) further observed that gastrointestinal disorders were more prevalent than neurological issues in dogs affected by lead poisoning.

Antemortem tests such as radiology, endoscopy, and surgery are commonly used to identify dogs with gastroduodenal ulceration or perforation and to describe their co-morbidities, response to treatment and outcome. The diagnosis of gastric ulceration relies on the signalment and medical history, as there is no specific breed, age or sex predispositions for the disease. The history may indicate NSAID therapy, ingestion of corrosive substances (lead in the present case), or other systemic diseases like hepatopathy, tumors or secondary metastases.

A combination of diagnostic modalities, including radiography, ultrasonography and endoscopy, is used to confirm the presence of gastric ulceration. Radiography (plain and contrast) is beneficial in determining potential causes of abdominal pain, such as gastric foreign bodies or radio-opaque substances like lead (Talbert *et al*., 2012). Barium contrast is a better option for detecting foreign bodies and for differential diagnosis, though small ulcers or larger defects filled with blood or debris may not be seen. Additionally, barium contrast studies are avoided in animals with suspected GI perforation due to the risk of barium extravasation exacerbating peritonitis (Ko and Mann, 2014).

Ultrasonography is a non-invasive technique used to detect gastric ulceration in dogs, typically revealing localised gastric wall thickening, crater-shaped fluid accumulation and reduced gastric motility. Ulcer craters are identified as mucosal defects with microbubbles. However, ultrasound is unable to differentiate between benign and malignant ulcers and also gas within the gastric lumen can obstruct imaging (Parrah *et al*., 2013). In this study, the ultrasonographic changes were primarily localised in the pyloric region of the stomach.

Endoscopic examination offers direct visualisation of the gastric mucosa, enabling the identification of lesions and facilitating the collection of biopsies for histopathological analysis. Endoscope-guided biopsy of the gastric mucosa typically reveals blunting, atrophy and cystic dilation of gastric glands, connective tissue proliferation, edema and blood vessel congestion in the submucosa. Biopsies are collected from the edge of the ulcer to rule out neoplasia, avoiding the ulcer bed centre to prevent perforation and renewed bleeding (Parrah *et al*., 2013).

Successful treatment of gastric ulcers relies on early recognition. Standard medical therapy includes identifying and removing the potential cause (lead in this case), antibiotics to prevent secondary bacterial infection, antacids, H2-blockers, proton-pump inhibitors and mucosal binders like sucralfate. Fluid therapy is crucial in dehydrated patients with gastric ulcers to maintain mucosal perfusion. Cytoprotective drugs such as sucralfate protect ulcerated tissue and should be administered one to two hours before histamine antagonists (Wada *et al*., 1997). Dietary fibres have demonstrated significant potential in binding heavy metal ions, such as lead (Wang *et al*., 2016). Specifically, psyllium seed (*Plantago ovata*), a natural, non-fermented gel-forming fibre (Gibb *et al*., 2023), has been demonstrated to assist in chelation and promote lead excretion (Basiri *et al*., 2020). The oral administration of psyllium significantly alleviated the pathological lesions in the gastrointestinal tract and other affected organs caused by lead, proving effective in the treatment of this case.

Therefore, this case highlights that gastric ulcers in dogs can arise from various causes including lead pellets. Employing a range of diagnostic methods, including clinical examination, imaging, endoscopy and histopathology of gastric mucosa, enables accurate identification of the underlying etiology and facilitates prompt intervention to prevent further complications.

**Summary**

Gastric ulcers in dogs, particularly those complicated by lead toxicity, require a comprehensive diagnostic approach involving radiography, ultrasonography and endoscopy for accurate assessment. Effective treatment includes removing the causative agent, such as lead, and employing a combination of proton pump inhibitors, antibiotics and mucosal protectants, along with dietary modifications like psyllium supplementation. This case demonstrates the critical role of integrated diagnostic and therapeutic strategies in managing chronic gastric ulcers and achieving successful outcomes.

**Conflict of interest**

The authors declare that they have no conflict of interest.

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