

Unusual eosinophilic gastritis with endoscopic examination in a cat

Mehmet Fatih Bozkurt¹, Erkan Yunus Tüfekci¹

¹Department of Veterinary Pathology, Faculty of Veterinary Medicine, Afyon Kocatepe University, Afyon/Türkiye

Key Words:

endoscopy
feline gastritis
gastric biopsy
gastritis
polyps

Received : 30.05.2023
Accepted : 21.09.2023
Published Online : 31.12.2023
Article Code : 1306076

Correspondence:
EY. TÜFEKÇİ
(erktuf@gmail.com)

ORCID
MF. BOZKURT : 0000-0002-1669-0988
EY. TÜFEKÇİ : 0000-0003-0722-1132

ABSTRACT

Eosinophilic gastritis is a rare type of gastritis seen in cats and dogs, and it is still not completely understood. The patient was an 8-year-old Persian cat presenting with increasingly chronic vomiting. An endoscopic visualization of the stomach was performed after a physical examination, blood tests and imaging. During the endoscopic examination of the stomach, nodular pseudo-polyps diffusely disturbed in the antrum region and protruding toward the lumen were macroscopically observed. After examining the biopsy samples taken from these structures, the patient was diagnosed with eosinophilic gastritis.

INTRODUCTION

In cats and dogs, as in other mammals, the inflammatory disease of the stomach in its simplest form is called gastritis. It is classified as acute or chronic depending on the occurrence of inflammation. Gastritis is usually clinically associated with vomiting, dehydration, and metabolic acidosis. Hemorrhage, edema, excessive mucus secretion, abscesses, granulomas, foreign bodies or their penetration, parasites, various inflammatory reactions, erosions, and ulcers cause inflammatory reactions on the gastric mucosal surface. The etiology of eosinophilic gastritis is not fully understood. It is characterized by a diffuse eosinophilic infiltration in the distal part of the stomach (Neiger, 2010). Although eosinophilic gastritis is uncommon in domestic animals, there have been some cases in pet carnivores. Eosinophilic gastritis can be classified into three headings. In the first type, ingestion of parasite larvae may result in the occurrence of polyp-like hyperplasia in the stomach and intestinal epithelium, which sometimes cause pyloric obstruction. Hypersensitivity reactions can be observed in a second type in which eosinophils spread more diffusely by unknown trigger antigens. Peripheral eosinophilia occurs mostly in this type. Eosinophilic infiltration, which presents itself, especially in the small intestine, is identified as eosinophilic gastroenteritis. The third type is scirrhous eosinophilic gastritis of unknown causes in cats and dogs. Microscopically, lesions in eosinophilic gastritis appear as large areas of eosinophil infiltration in the mucosa and submucosa (Gelberg, 2017). Eosinophilic gastritis is usually reactions caused by food allergens. Although mucosal thickening is sometimes identified during ultrasound examination, clinical findings are not pathognomonic. Endoscopic

biopsy applications can be advantageous in terms of duration of the procedure and damage to the tissue when compared to biopsy samples taken by laparoscopic methods. However, biopsies taken with endoscopic methods will remain superficial and may not provide information about mucosal lesions. The purpose of this study is to examine pseudopolyps found in the stomach cytologically and histopathologically.

MATERIALS and METHODS

An 8 year old, castrated, male Persian cat with increasing complaints of chronic vomiting was admitted to an animal hospital. According to its clinical history, it was an indoor cat, and it was fully vaccinated and had completed anti-parasitic treatments. The initial vomiting frequency was once or twice a month but eventually it began to occur every day. The patient had no complaints other than vomiting.

Blood, serum biochemistry, urine and hormone test conducted after the physical examination did not reveal any abnormalities that could cause vomiting. Hematology results were (Idexx Procyte Dx Hematology Analyser) RBC 9 (6.54-12.20) M/ μ L, HCT 37.9 (30.3 - 52.3)%, hemoglobin 12.8 (9.8 - 16.2) g/dl, MCV 42.1 (35.9 - 53.1) fL, MCH 14.2 (11.8 - 17.3) pg, MCHC 33.8 (28.1-35.8) g/dL, reticulocytes 9.9 (3-50) K/ μ L, WBC 8.69 (2.87-17.02) K/ μ L, neutrophils 4.86 (1.48 - 10.29) K/ μ L, lymphocytes 2.5 (0.92 - 6.88) K/ μ L, monocytes 0.29 (0.05 - 0.67) K/ μ L, eosinophils 0.94 (0.17 - 1.57) K/ μ L, basophils 0.10 (0.01 - 0.26) K/ μ L, and plateletes 285 (151 - 600) K/ μ L. Blood chemistry test (Idexx Catalyst One Chemistry) results were as follows: glucose 84 (74 - 159) mg/

dL, creatinine 1.2 (0.8 - 2.4) mg/dL, urea 15 (16 - 36) mg/dL, SDMA 9 (0 - 14) µg/dL, UPC < 0.08 (non-proteinuric), BUN: creatinine ratio 12, phosphorus 4.3 (3.1 - 7.5) mg/dL, calcium 9 (7.8 - 11.3) mg/dL, total protein 6.4 (5.7 - 8.9) g/dL, albumin 2.7 (2.2 - 4.0) g/dL, globulin 3.7 (2.8 - 5.1) g/dL, albumin: globulin ratio 0.7, ALT 57 (12 - 130) U/L, ALP 29 (14 - 111) U/L, GGT 0 (0 - 4 U/L), total bilirubin < 0.1 (0.0 - 0.9) mg/dL, cholesterol 96 (65 - 225) mg/dL tT4 1.2 (0.8 - 4.7) µg/dL. Feline pancreas-specific lipase enzyme was in the normal range (Idexx Snapshot Dx Analyzer). A urine sample collected via cystocentesis was tested with a urine strip (Idexx Vetlab UA) and gave the following results: color, pale yellow, sg: 1.043,

pH:6.5, urine protein; negative; glucose, negative; ketones, negative; blood/hemoglobin 50 ery/µL; bilirubin, negative; and urobilinogen: 1 µg/dL. A blood smear was performed to evaluate eosinophils. Correlations between blood smear results and the blood count determined.

On sonographic examination, a slight thickening of the muscular layer of the small intestine and 0.3 - 1 cm diameter cystic structures were observed in the right and left kidneys.

The decision was made for endoscopic imaging due to the lack of results indicating any factor that would induce vomit-

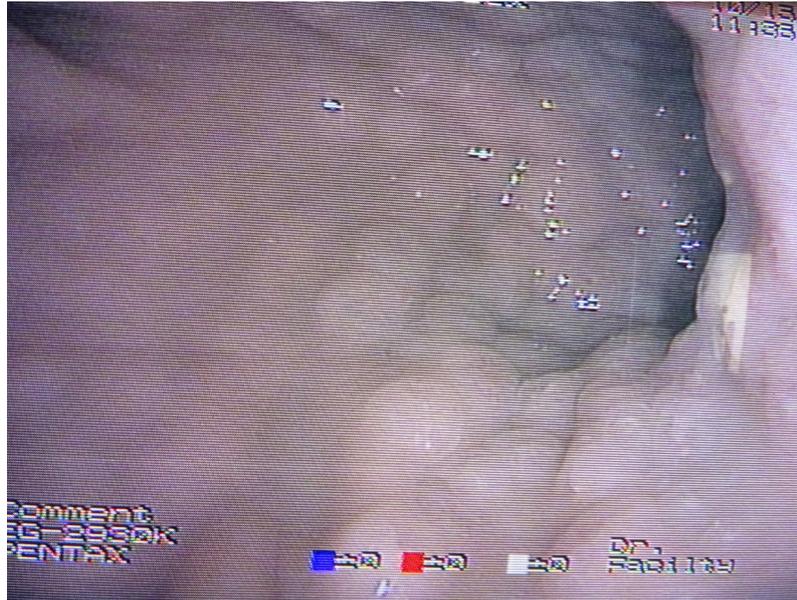


Figure 1. Diffusely distributed pseudo-polyps in the pyloric antrum. Biopsies were taken from this part.

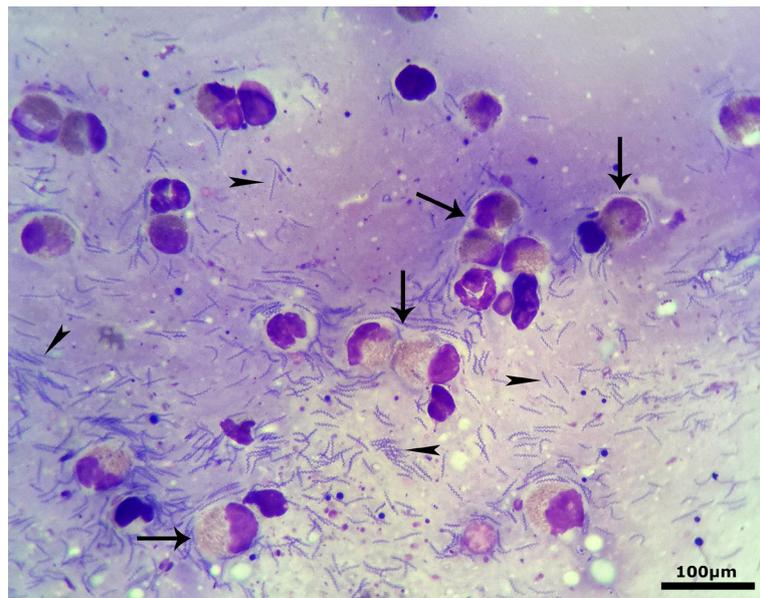


Figure 2. A cytological specimen obtained from the gastric antrum. High numbers of eosinophils (arrows) and spiral shaped bacteria (arrowheads) are shown (Wright's stain; bar 100 µm).

ing.

No macroscopic anomalies from the patient's mouth to the entrance of the stomach, including the esophagus, were found in the endoscopic examination with a video gastroscope (Pentax EG-2930K). Diffusely distributed pseudo polyps developing toward the lumen of the stomach were detected in

Vomiting ceased shortly after antibiotic (amoxicillin-clavulanate), antiemetic (maropitant) and H2 receptor antagonist (ranitidine) treatment had commenced. The diet switched to hydrolyzed protein containing food. Endoscopic examination of the stomach was not repeated due to the lack of further clinical symptoms.

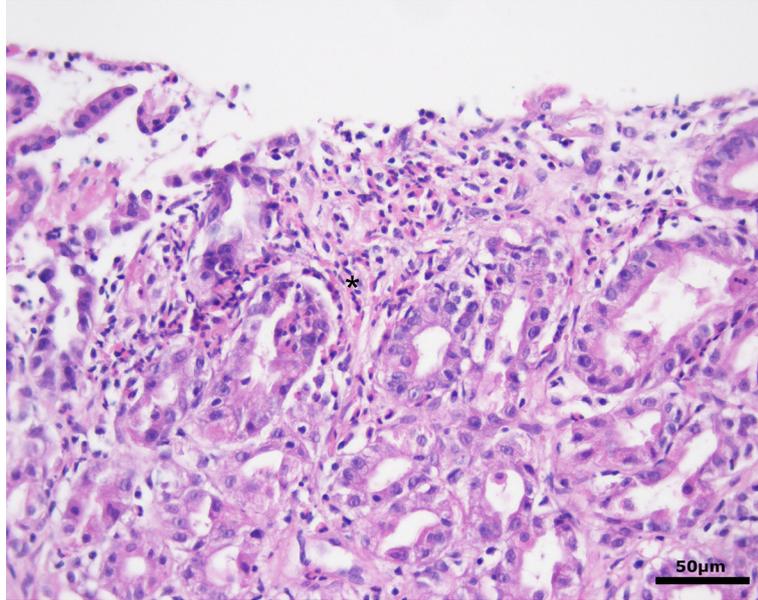


Figure 3. Numerous eosinophils are widespread within lamina propria. Mild atrophy is seen in the glands(asterisk) (Hematoxylin and eosin stain; 50 μ m).

the pyloric antrum (Figure 1). These foci were approximately 2 - 3 mm in size. Biopsies were taken from the structures in the pylorus region under endoscopic guidance. The biopsies were cytologically stained using Wright's staining method. Increased number of eosinophils was detected on a slightly eosinophilic background in cytologically examined samples under a light microscope (Figure 2). Eosinophil leukocytes were followed by neutrophil leukocytes and lymphocytes in terms of density. Spiral shaped bacteria become blue when stained with Wright's eosin methylene blue (Figure 2).

Biopsies were fixed in a 10% formalin solution and embedded in paraffin. 4 μ m in thickness were stained with hematoxylin-eosin. Histopathological examination revealed a large amount of degranulated eosinophils in the lamina propria close to the lumen (Figure 3). According to the histopathological classification specified by the World Small Animal Veterinary Association (WSAVA) Gastrointestinal Standardization Group, the eosinophil leukocyte count should moderate, from 50 up to 100 at high magnification; 100 indicating a marked increase. In our sample, eosinophil leukocytes were markedly over 100 and scattered over wide areas of the lamina propria (Day et al. 2008). The number of eosinophil leukocytes in this area in the normal gastric mucosa is accepted to be between 0 and 2 at high magnification (Washabau et al. 2010). Mild atrophy was observed in the glands and extensive spiral-shaped bacteria were detected (Figure 3).

DISCUSSION

Eosinophilic gastritis is rare, and its etiology is not fully understood (Gelberg 2017; Votto et al. 2021). No laboratory findings and imaging methods, other than endoscopic imaging and biopsy examination, produce that lead to a diagnosis. Peripheral blood eosinophilia was found in approximately 80% of patients in human studies (Gonsalves, 2019). In our case we did not find a correlation between peripheral eosinophils and gastric inflammation. A diagnosis could only be reached by imaging the nodular pseudo polyps and evaluating the patient's general condition with histopathological and cytological examination of biopsies taken from the nodular pseudo polyps. Studies on eosinophilic gastritis in humans indicate two types of lesions: those showing nodular structure; or those erosional structures. The difference between nodular or ulcerative is morphological. Both types are variations of the same inflammatory response. However, some cases of eosinophilic gastritis do not conform to the two aforementioned types (Sato et al., 2017). A literature review failed to find mention of macroscopic imaging of pseudo polyps as nodules in veterinary medicine, even though it has previously been described in human medicine (Fisher et al. 2017; Sato et al. 2017). We believe that this type of imaging of gastric polyps we performed is a first in veterinary medicine. The ulceration of the diseased mucosa and flow of plasma proteins into the lumen in eosinophilic gastritis has been mentioned in veterinary medicine (Neiger, 2010).

Histopathological examination remains important despite the development of modern diagnostic methods (Rugge et al., 2020). The WSAVA has standardized it in order to ensure that gastric biopsies examined by pathologists are classified according to a certain standard. In this study, inflammation was interpreted as normal, mild, moderate, or severe depending on the number of leukocytes in a given area (Day et al., 2008). The high amount of eosinophilic leukocytes detected in the cytological examination of the endoscopic biopsy specimen in parallel with the histopathological examination may be sufficient for diagnosis. Since eosinophilic gastritis is usually limited to the mucosal layer (Neiger, 2010), endoscopic biopsy applications are thought to be sufficient in the diagnosis of eosinophilic gastritis without the need to take full-thickness biopsy samples. Thus, when taking the patient into consideration, anesthesia time, operation stress, and post-op care will not be necessary.

In previous human studies, some cases of gastritis were due to *Helicobacter pylori* causing an increase in the number of eosinophils in the stomach. In humans *H. pylori* is one of the most common causes of gastrointestinal diseases (Aydemir et al., 2004). In contrast to humans, in cats, *H. heilmannii* and *H. felis* are more frequently identified than *H. pylori* (Neiger et al., 1998). *H. heilmannii* can even be found in healthy cats' stomachs and may cause minimal inflammation (Norris et al., 1999). However, these species cannot be differentiated using only a light microscope (Neiger et al., 1998). Human-based studies have suggested that cobblestone nodules or polyps may be seen in eosinophilic gastritis (Connors, 2007).

We believe that a *Helicobacter* species caused the eosinophilic gastritis and the pseudo polyps in our case. Imaging and biopsy examinations of the stomach are recommended for long-term complaints that do not respond to symptomatic treatments. This type of gastritis might be more frequently encountered if there is an increase in the performance of endoscopic interventions on patients with such findings. Cytological examination of biopsy samples taken from lesions and mucosa will accelerate the diagnosis and, consequently, the treatment of some diseases.

DECLARATIONS

Ethics Approval

Not applicable.

Conflict of Interest

The authors declare that they have no competing interests

Consent for Publication

Not applicable.

Author contribution

Idea, concept and design: MFB, EYT

Data collection and analysis: MFB, EYT

Drafting of the manuscript: EYT, MFB

Critical review: MFB, EYT

Acknowledgements

We would like to thank VTM Animal Hospital, Ankara, Turkey for their assistance.

REFERENCES

- Aydemir, S. A., Tekin, I. O., Numanoglu, G., Borazan, A., & Ustundag, Y. (2004). Eosinophil infiltration, gastric juice and serum eosinophil cationic protein levels in *Helicobacter pylori*-associated chronic gastritis and gastric ulcer. *Mediators of inflammation*, 13(5-6), 369–372. <https://doi.org/10.1155/S0962935104000559>
- Connors, J. M. (2007). Eosinophilic Gastritis. In: S.J. Enna, & D.B. Bylund (Eds.). *XPharm: The Comprehensive Pharmacology Reference* (pp. 1–6). Elsevier. <https://doi.org/10.1016/b978-008055232-3.60718-4>
- Day, M. J., Bilzer, T., Mansell, J., Wilcock, B., Hall, E. J., Jergens, A., Minami, T., Willard, M., Washabau, R., & World Small Animal Veterinary Association Gastrointestinal Standardization Group (2008). Histopathological standards for the diagnosis of gastrointestinal inflammation in endoscopic biopsy samples from the dog and cat: a report from the World Small Animal Veterinary Association Gastrointestinal Standardization Group. *Journal of comparative pathology*, 138 Suppl 1, S1–S43. <https://doi.org/10.1016/j.jcpa.2008.01.001>
- Fisher, A., Edhi, A., Desai, T., & Khurram, D. (2017). Eosinophilic Gastroenteritis: More Common Than we Think and Perhaps Ominous in Women. *American Journal of Gastroenterology*, 112, S267–S270. <https://doi.org/10.14309/00000434-201710001-00510>
- Gelberg, H. B. (2017). Alimentary System and the Peritoneum, Omentum, Mesentery, and Peritoneal Cavity. *Pathologic Basis of Veterinary Disease*, 324-411.e1. <https://doi.org/10.1016/b978-0-323-35775-3.00007-2>
- Gonsalves, N. (2019). Eosinophilic Gastrointestinal Disorders. *Clinical Reviews in Allergy & Immunology*, 57(2), 272–285. <https://doi.org/10.1007/s12016-019-08732-1>
- Neiger, R. (2010). Diseases of the Stomach. In: J. M. Steiner (Ed.). *Small Animal Gastroenterology* (pp. 162) Schlütersche.
- Neiger, R., Dieterich, C., Burnens, A., Waldvogel, A., Corthésy-Theulaz, I., Halter, F., Lauterburg, B., & Schmassmann, A. (1998). Detection and Prevalence of *Helicobacter* Infection in Pet Cats. *Journal of Clinical Microbiology*, 36(3), 634–637. <https://doi.org/10.1128/jcm.36.3.634-637.1998>
- Norris, C. R., Marks, S. L., Eaton, K. A., Torabian, S. Z., Munn, R. J., & Solnick, J. V. (1999). Healthy Cats Are Commonly Colonized with “*Helicobacter heilmannii*” That Is Associated with Minimal Gastritis. *Journal of Clinical Microbiology*, 37(1), 189–194. <https://doi.org/10.1128/jcm.37.1.189-194.1999>
- Rugge, M., Sugano, K., Sacchi, D., Sbaraglia, M., & Malfertheiner, P. (2020). Gastritis: An Update in 2020. *Current Treatment Options in Gastroenterology*, 18(3), 488–503. <https://doi.org/10.1007/s11938-020-00298-8>

Sato, M., Shoda, T., Shimizu, H., Orihara, K., Futamura, K., Matsuda, A., Yamada, Y., Irie, R., Yoshioka, T., Shimizu, T., Ohya, Y., Nomura, I., Matsumoto, K., & Arai, K. (2017). Gene Expression Patterns in Distinct Endoscopic Findings for Eosinophilic Gastritis in Children. *The Journal of Allergy and Clinical Immunology: In Practice*, 5(6), 1639-1649.e2. <https://doi.org/10.1016/j.jaip.2017.03.030>

Votto, M., De Filippo, M., Olivero, F., Raffaele, A., Cereda, E., De Amici, M., Testa, G., Marseglia, G. L., & Licari, A. (2020). Malnutrition in Eosinophilic Gastrointestinal Disorders. *Nutrients*, 13(1), 128. <https://doi.org/10.3390/nu13010128>

Washabau, R. J., Day, M. J., Willard, M. D., Hall, E. J., Jergens, A. E., Mansell, J., Minami, T., & Bilzer, T. W. (2010). Endoscopic, Biopsy, and Histopathologic Guidelines for the Evaluation of Gastrointestinal Inflammation in Companion Animals. *Journal of Veterinary Internal Medicine*, 24(1), 10–26. <https://doi.org/10.1111/j.1939-1676.2009.0443.x>